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# THE AMERICAN JOURNAL OF PROCTOLOGY

*OFFICIAL PUBLICATION OF THE INTERNATIONAL ACADEMY OF PROCTOLOGY*

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**The General-Adaptation-Syndrome and the Gastrointestinal Diseases of Adaptation**

**Reetosigmoidal Polyps**

**Perinanal Apocrine Gland Fistula**

**Surgical Lesions of the Right Half of the Colon and Their Treatment**

**Pruritus Ani**

**Editorials • Book Reviews • Annual Index**

**VOLUME 2** **NUMBER 4**

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*The First Authoritative Journal of Proctology and Allied Subjects*

VOLUME 2 DECEMBER, 1951 NUMBER 4

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THE AMERICAN JOURNAL OF

# PROCTOLOGY

OFFICIAL PUBLICATION

*of the*

INTERNATIONAL ACADEMY of PROCTOLOGY

1819 Broadway, New York 23, N. Y.

Editorial Office: 43-55 Kissena Blvd., Flushing, N. Y.

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THE AMERICAN JOURNAL OF
PROCTOLOGY
*The First Authoritative Journal of Proctology and Allied Subjects*

VOLUME 2 DECEMBER, 1951 NUMBER 4

# THE GENERAL-ADAPTATION-SYNDROME AND THE GASTROINTESTINAL DISEASES OF ADAPTATION

HANS SELYE, M.D.*
Montreal, Canada

At the invitation of the editor of THE AMERICAN JOURNAL OF PROCTOLOGY I should like to present, in the following pages, a brief synopsis of the concept of the general-adaptation-syndrome, as well as a summary of the most important recent contributions to the understanding of those maladies which we interpret as being primarily gastrointestinal "diseases of adaptation". A much more detailed, systematic description of the pertinent data published prior to 1950, has been given in our treatise "Stress" [121], which appeared last year. Hence, here, we shall limit ourselves almost entirely to a consideration of the most recent contributions to this field, in order to furnish the interested proctologist with a guide to the current literature. Papers of an earlier date will only be considered inasmuch as they furnish data essential to the understanding of the adaptation-syndrome concept in general.

## THE GENERAL-ADAPTATION-SYNDROME (G-A-S)

*The concept of stress:*—By a series of experiments on animals I could show, in 1936, that the organism responds in a stereotypical manner to a variety of widely different agents, such as: infections, intoxications, trauma, nervous strain, heat, cold, muscular fatigue or x-irradiation. The specific actions of all these agents are quite different. Their only common feature is that they place the body in a state of general (systemic) stress. Hence, we concluded that the stereotypical response—which is superimposed upon all specific effects—represents the somatic manifestations of nonspecific "stress" itself.

But what is *nonspecific "stress"?* The term had long been used in physics to denote the results of the interaction between a force and the resistance opposed to it. For instance, pressure and tension cause stress in inanimate matter. I thought that the above mentioned nonspecific response represents the biologic equivalent of such physical stress. And the term has now been quite generally accepted in this sense, not only in English, but—since attempts to translate "stress" led to much confusion—also in most other languages.

*The concept of the G-A-S:*—The most outstanding manifestations of this stress-response were: *adrenocortical enlargement* with histologic signs of hyperactivity, *thymicolymphatic involution* with certain concomitant changes in the blood-count (eosinopenia, lymphopenia, polynucleosis) and *gastrointestinal ulcers,* often accompanied by other manifestations of *damage or "shock".*

We were struck by the fact that, while during this reaction all the organs of the body show involutional or degenerative changes, the adrenal cortex actually

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This investigation was made possible by a grant of the National Research Council of Canada.

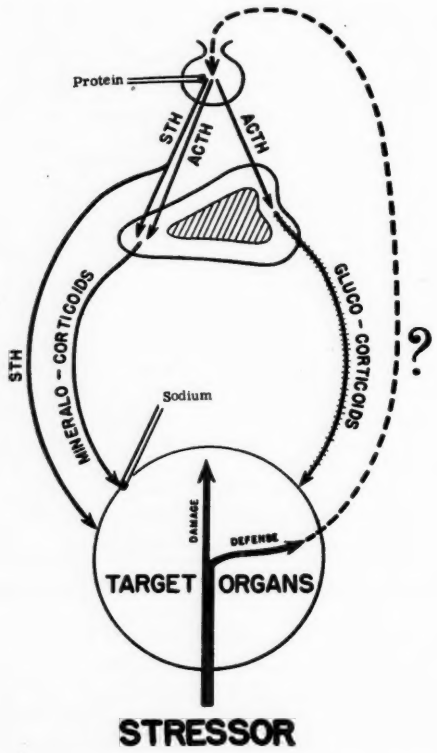


Fig. 1—*Schematic diagram illustrating the principal interrelations between the hypophysis, the adrenal cortex and the peripheral target organs during the General-Adaptation-Syndrome.* The *stressor* (trauma, infection, burns, etc.) acting directly upon the cells produces *damage.* At the same time, it also mobilizes *defense* by evoking a stimulus, which induces the anterior pituitary to produce ACTH; under certain circumstances it may also cause a discharge of STH. The nature of this *first mediator* between the directly injured organ and the anterior pituitary is not yet known (humoral, nervous?). Hence, here it is indicated merely by an interrupted line, labelled with a question mark. ACTH induces the adrenal cortex to produce predominantly *glucocorticoid* compounds, whose effect upon the response of the various target organs is generally inhibitory (e.g., catabolism, diminution of granuloma formation and of allergic responses, diminished resistance to infection). Conversely, STH enhances a variety of defensive reactions in the target organs (e.g., anabolism, augmentation of granuloma formation and of allergic responses, increased resistance to infection), primarily by stimulating the connective tissue. Part of this action is undoubtedly not mediated through the adrenal cortex, but this direct effect sensitizes the connective tissue elements to the (essentially similar) actions of the *mineralocorticoids.* It is probable that STH also acts by increasing the production of mineralocorticoids. However, in itself, it cannot maintain the cortical cells in a responsive condition, hence its "corticotrophic" effect is dependent upon the simultaneous availability of ACTH. In the final analysis the physiologic and pathologic responses of the target organs to stressor agents largely depend upon the balance between the mineralocorticoids and STH on the one hand, and ACTH and the glucocorticoids on the other.
The production of corticotrophic hormone by the pituitary is enhanced by high *protein* diets, while the actions of mineralocorticoids upon most target organs is augmented by excess *sodium.* Thus dietary factors *"condition"* the response of the body to hormones produced during stress.

seems to flourish on stress. We suspected this adrenal response to play a useful part in a nonspecific adaptive reaction, which we visualized as a "call to arms" of the body's defense forces and named the "alarm-reaction" [114].

Subsequent studies showed us that the alarm-reaction is but the first stage of a much more prolonged *General-Adaptation-Syndrome* (G-A-S). The latter comprises three distinct stages, namely:

(1) the *alarm-reaction* (A-R), in which adaptation has not yet been acquired,

(2) the *stage of resistance* (S-R), in which adaptation is optimal,

(3) the *stage of exhaustion* (S-E), in which the acquired adaptation is lost again.

*The mechanism of the G-A-S:*—In order to elucidate the kinetics of this syndrome we proceeded as follows:

Rats were *adrenalectomized* and then exposed to stressor agents. This showed us that in the absence of the adrenals stress can no longer cause thymicolymphatic involution or characteristic blood-count changes.

When adrenalectomized rats were treated with the impure *cortical extracts* available at that time, it became evident that thymicolymphatic involution and the typical blood-count changes could be produced by adrenal hormones even in the absence of the adrenals. The latter, therefore, were considered to be indirect results of stress mediated by corticoids.

Conversely, the gastrointestinal ulcers and other manifestations of pure damage were actually more severe in adrenalectomized than in intact animals and could be lessened by treatment with cortical extracts. It was concluded that these lesions are not mediated through the adrenal and are combatted by an adequate adrenocortical response to stress[113].

But what stimulates adrenocortical function during stress? In 1937, we found that among many surgical interventions tried, only *hypophysectomy* prevents the adrenal response during the alarm-reaction. Hence, we concluded that stress stimulates the cortex through an adrenocorticotrophic hormone, now known as ACTH[115].

Then pure cortical steroids became available, thanks to the classical investigation of Kendall and Reichstein. With these, we could show that administration of *mineralocorticoids, or M-Cs* (such as desoxycorticosterone) produces experimental replicas of the so-called *hypertensive and rheumatic diseases;* notably, *nephrosclerosis, hypertension, vascular lesions* (especially periarteritis nodosa and hyalin necrosis of arterioles)[123], as well as *arthritic* changes resembling, in acute experiments, those of rheumatic fever, and after chronic treatment, those of rheumatoid arthritis[129]. Yet, even very high doses of mineralocorticoids did not induce any noteworthy thymicolymphatic or blood-count changes.

Significantly, exposure of animals to nonspecific stressor agents (e.g., cold) produced marked adrenocortical enlargement and organ changes very similar to those elicited by the administration of mineralocorticoids[116].

*Gluco-corticoids or G-Cs* (such as cortisone), on the other hand, were highly potent in causing *thymicolymphatic* involution and in eliciting the characteristic *blood-count* changes of the alarm-reaction. They also tended to inhibit the hypertensive and rheumatic changes which can be elicited in animals by mineralocorticoids. Thus, in many respects, the two types of corticoid hormones antagonize each other[118, 121].

*Inflammatory granulomas,* especially those produced in the vicinity of joints by the local application of irritants (e.g., formalin, mustard powder), as well as cer-

tain allergic reactions, are likewise aggravated by mineralo- and prevented by gluco-corticoids. Apparently, the response of the adrenal cortex is most important not only in defense against systemic stress (affecting the whole organism), but also in the manifold topical defense reactions which occur upon exposure to *local stress* (e.g., bacterial or chemical irritants, responses of a "shock organ" to an allergen)[119, 121].

*Certain crude anterior-pituitary extracts*[117, 118], duplicate the above mentioned actions of M-Cs upon the cardiovascular system, the blood-pressure and the kidneys. The hypophyseal preparations which we used, were definitely corticotrophic,

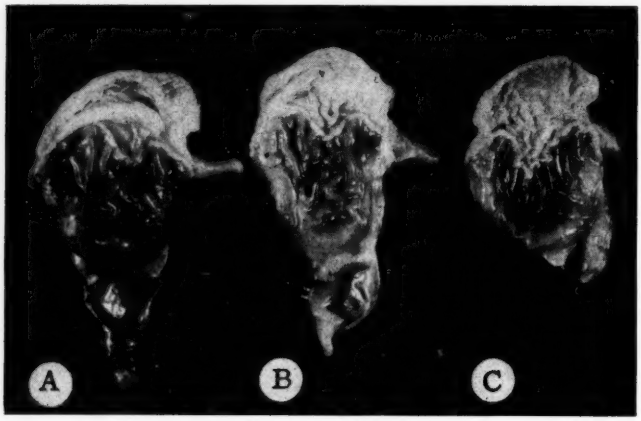


Fig. 2—*Gastric ulcers during the A-R, after "conditioning" by intravenous injection of India ink. A.* Intense hemorrhagic ulcer formation in the stomach of a rat which received an intravenous injection of India ink during an A-R produced by exposure to cold. Eight rats similarly treated all showed gastric ulcers of this intensity.

*B.* Gastric mucosa of a rat exposed to cold in the same manner as that whose stomach is illustrated in A. Although this animal showed the most intense gastric lesions in a series of eight rats similarly treated, only two or three minute gastric erosions are visible.

*C.* Gastric mucosa of a rat treated with India ink, in the same manner as that whose stomach is shown in A, but not exposed to cold. There is no trace of gastric ulcer formation. The mechanism through which India ink produces this effect has not yet been elucidated, but perhaps the ink particles act by interfering with the nutrition of the gastric mucosa through the formation of microembolisms or by diapedesis across damaged vessels. In any event the experiment clearly demonstrates the possibility of "selective conditioning" of one target organ to a typical A-R lesion. The other manifestations of the A-R (e.g., adrenal enlargement, thymus atrophy) were not noticeably aggravated by the India ink injection. (Selye and Guillemin, unpublished.)

in that they enlarged the adrenal cortex, but they were particularly rich in the so-called "growth hormone" or *somatotrophic hormone* (STH). As soon as we were able to obtain purified ACTH, it became evident that the above mentioned pathogenic actions of the crude anterior-pituitary preparations could not be due to their ACTH content, since even the highest tolerable doses of the latter hormone failed to duplicate their predominant M-C effects. On the other hand, overdosage with pure STH caused cardiovascular and renal lesions, identical with those previously observed in animals treated with M-Cs. It was concluded that the above mentioned actions of our crude anterior-pituitary preparations were mainly due to their STH content. It remains to be seen to what extent STH acts indirectly

by stimulating the M-C production of the adrenal cortex, or directly by sensitizing the peripheral tissues to M-Cs. Preliminary observations suggest that both these mechanisms may be implicated[122].

From the internists' point of view perhaps the most interesting rôle of STH in the Adaptation Syndrome is that it can effectively *combat catabolism and sensitivity of infections.* Animals heavily overdosed with ACTH or G-Cs, tend to lose a great deal of weight. Eventually they die, almost always as a result of generalized septicemia, caused by normally sacrophytic microorganisms. In rats the lung tissue appears to be singularly predisposed to such infections. Under these conditions, adequate doses of STH prevent the loss of body weight as well as the excessive microbial proliferation[125]. It remains to be seen to what extent these actions of STH will prove to be of value in the management of infections in man.

*Conditioning of hormone actions:*—As work along these lines progressed it became increasingly obvious that the activity of the hormones produced during stress depends largely upon a variety of "conditioning factors". Both the

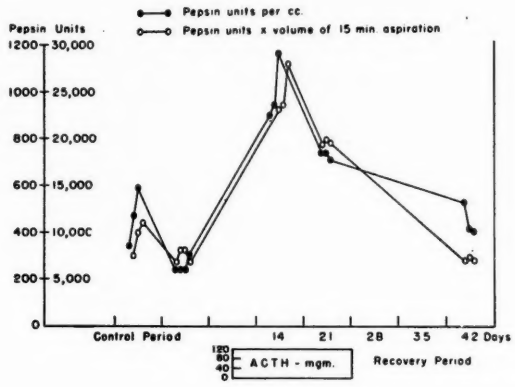


Fig. 3—*Effect of ACTH upon gastric pepsin.* Marked increase in pepsin concentration and total pepsin secretion with a return to the control levels upon discontinuation of ACTH. (*Courtesy of Drs. S. J. Gray, J. A. Benson, Jr., R. W. Reifenstein and H. M. Spiro.*)

production of the "stress hormones" and their effect upon individual target organs proved to be greatly influenced by heredity, previous exposure to stress, the diet, etc. Thus, for instance, the production of corticotrophic hormone by the pituitary is enhanced by a high-protein diet, while the action of M-Cs upon most target organs is augmented by excess sodium.

In the final analysis, such factors could actually determine whether exposure to stress would be met by a physiologic G-A-S or cause Diseases of Adaptation; indeed, in the latter instance, they appear to be responsible for the selective breakdown of one or the other organ. We felt that this might explain why the same kind of stress may cause diverse types of "Diseases of Adaptation" in different individuals.

*The concept of the Diseases of Adaptation:*—From our above mentioned experiments, we concluded that the pathogenicity of many systemic and local irritants depends largely upon the function of the hypophysis-adrenocortical system. The latter may either enhance or inhibit the body's defense reactions against

stressor agents. We think that derailments of this adaptive mechanism are the principal factor in the production of certain maladies which we consider, therefore, to be essentially *Diseases of Adaptation.*

Among the *derailments of the G-A-S which may cause disease,* the following are particularly important:

(1) An *absolute excess* or deficiency in the amount of corticoids and STH produced during stress.

(2) A *disproportion* in the relative secretion, during stress, of ACTH and G-Cs on the one hand, and of STH and M-Cs on the other.

(3) Production by stress of metabolic derangements, which abnormally alter the *target organ's response* to STH, ACTH or corticoids (through the phenomenon of "conditioning").

(4) Finally, we must not forget that, although the hypophysis-adrenal mechanism plays a prominent rôle in the G-A-S, *other organs* which participate in the latter (e.g., nervous system, liver, kidney) may also respond abnormally and become the cause of disease during adaptation to stress.

## Gastrointestinal System

*Stressors:*—Studies on the so-called "postoperative disease" in man led to the conclusion that it corresponds to the initial phases of the G-A-S. Although most of the characteristic manifestations of the A-R develop as a result of *trauma,* hemorrhagic erosions of the gastric mucosa are comparatively rare[94]. Yet, allegedly, even a typical peptic ulcer can ensue after a single trauma in man[86].

The acute ulcers of the upper intestinal tract, which tend to occur after *burns* in man, are usually localized in the duodenum, rarely in the gastric mucosa. They are commonly known as "Curling's ulcer" [3, 27, 40, 66, 71, 98, 139]. Occasionally, burns may also cause hemorrhages from the esophagus[69].

In a large series of dogs with experimental third degree *burns,* 53 per cent exhibited typical Curling's ulcers[52], which resembled those seen in man.

Even burns which are not in themselves of sufficient intensity to produce gastroduodenal ulcers in the dog, elicit them in animals simultaneously treated with *histamine.* This has been largely ascribed to the concomitant hemoconcentration, since avoidance of the latter, by proper therapeutic measures, prevents the formation of gastric ulcers, even in dogs simultaneously treated with histamine and subjected to burns[38].

*Ionizing radiations* are particularly prone to produce gastrointestinal erosions. This was again clearly demonstrated by autopsies of atomic bomb victims[142].

While in the rat x-irradiation regularly produces acute bleeding gastric ulcers, this is much less commonly the case in the rabbit. Since other manifestations of the A-R (adrenocortical stimulation, thymicolymphatic involution) were elicited with equal readiness in rat and rabbit, it has been concluded that the latter species is selectively resistant to the gastric manifestations of the G-A-S[39].

Exposure to *cold* of sufficient severity to elicit the classic A-R syndrome, results in hemorrhagic erosions of the gastric mucosa in rats, but not in rabbits[39].

The effect upon the gastrointestinal tract of *nervous and emotional* stressors has again been the subject of numerous investigations. Direct observations (e.g., through gastric fistulae, sigmoidoscope) suggest that in man the secretion, contractility, and blood-supply of the stomach[87, 145] and colon[41, 43] are significantly influenced not only by the intensity, but also by the quality, of the emotional stimuli. Both constipation and diarrhea have been found to occur as direct consequences of emotional stress in man[24]. In the dog and monkey, gastric secretion can be selectively affected by frontal lobe stimulation[24]. All these findings illustrate

that the specific effects of stressors can considerably modify the response of the gastrointestinal tract during the G-A-S.

In the rat, intense light or noise can produce characteristic, A-R type, gastric erosions; at the same time, they decrease the resistance of the gastric mucosa to subsequent injections of histamine[81].

*Hypoxia* diminishes the propulsive motility of the small intestine in the rat, but this action gradually vanishes as the animal becomes adapted[138].

Very acute hypoxia may even produce appendicitis-like changes in the rat, concurrently with other characteristic manifestations of the A-R[35].

Toxic doses of *drugs* likewise cause characteristic A-R changes in the gastric mucosa. This has now been noted in rats treated with *nitrogen mustard*[39], and in man after fatal *insulin* intoxication[1]. Since it is invariably associated with other manifestations of the A-R, it has been interpreted as part of the G-A-S and not as the result of any specific pharmacologic action of insulin or the nitrogen mustard.

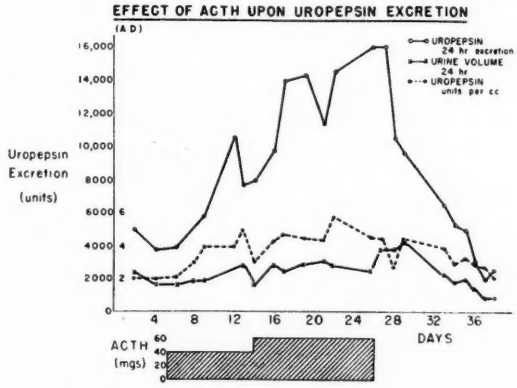


Fig. 4—*Effect of ACTH upon uropepsin excretion.* This patient died of a massive gastric hemorrhage and duodenal perforation following four courses of ACTH therapy for rheumatoid arthritis. During the first administration of ACTH the urinary uropepsin increased within 22 days to the exceedingly high range of 16,740 units, an unprecedented level for patients with active duodenal ulcer. (*Courtesy of Drs. S. J. Gray, J. A. Benson, Jr., R. W. Reifenstein and H. M. Spiro.*)

Barbiturates tend to prevent the development of various kinds (histamine, atophan) of experimental ulcers in the dog. This was taken as evidence showing the importance of purely neurogenic factors in the development of such lesions[59].

Our earlier observations, concerning the production in rats of an appendicitis-like condition by *histamine and formalin*, have now been confirmed. Here again, the change was ascribed to the A-R and not to any specific pharmacologic action of the drugs used[35].

If *India ink* is injected intravenously in the rat, during an A-R, produced by exposure to cold or excessive muscular exercise, the incidence of acute gastric ulcers is much greater than in similarly stressed controls receiving no India ink. Curiously, in rats in which an A-R was produced by repeated subcutaneous formaldehyde injections, it has not been possible to increase the incidence of these acute gastrointestinal erosions by India ink[48]. Histologic studies of the ulcers revealed numerous microemboli of India ink in the surrounding capillaries.

Several earlier investigators expressed the view that hemoconcentration and thromboembolic processes may increase the sensitivity of the gastrointestinal mucosa to acute ulcer formation, such as occurs, for instance, after burns[38]. It is probable that all these factors act largely by decreasing the vitality of the mucosa and thus diminishing its resistance against self-digestion.

*Phenyl-cinchoninic acid* is commonly used to produce experimental peptic ulcers in dogs. It has been pointed out that this compound is closely related to hydroxyphenyl-cinchoninic acid, which is currently recommended as an ACTH substitute for the treatment of "collagen diseases". Since, under certain conditions, ACTH likewise increases the tendency towards the formation of gastrointestinal ulcers, the possibility has been considered that both these cinchoninic acid derivatives may act through the discharge of ACTH[84].

Intoxication with large doses of *pilocarpin* produces ischemic lesions and predisposes to peptic ulceration of the upper gastrointestinal tract in the guinea-pig[11]. Since pilocarpin is an autonomic drug, this was considered to be the result of some nervous derangement; the possible participation of stress, as such, has not been considered.

In dogs with gastric pouches, the ability of histamine to stimulate gastric secretion is inhibited during fever produced by various *pyrogens*. In this study, no attempt was made to determine whether this is due to a specific effect of the toxic substances or to the resulting nonspecific stress[13].

*Tannic acid* intoxication causes typical manifestations of the G-A-S, in the rat. Hence, it has been surmised that the "hemorrhagic gastric erosions produced by parenterally administered tannic acid are a part of the syndrome present in the alarm-reaction" [72].

Severe *starvation* may produce gastroduodenal ulcers[63] or "hunger diarrhea" [137] in man. In the rat, inanition causes atrophy of the gastrointestinal mucosa with marked reduction of its digestive power. These experimental lesions have been compared to the "hunger diarrhea" of chronically starved patients[136], but the possible participation of the G-A-S has not been envisaged.

## Stimuli Influencing the G-A-S

*Anterior-lobe hormones:*—In dogs with "Mann-Williamson ulcers", ACTH appears to be of definite benefit, perhaps because it increases the production of an anti-ulcer factor "*uroanthelone*" [107]. On the other hand, in patients under ACTH therapy, intense gastric pain, exacerbation of preexistent ulcers, and perhaps even neoformation of perforating gastric ulcers may occur. These detrimental effects could be due to the fact that ACTH stimulates the production of *uropepsin, pepsin, gastric HCl* and the total amount of *gastric juice*[44, 49, 107].

*Adrenal hormones:*—The rate of secretion by the isolated stomach of the *adrenalectomized* mouse is essentially normal, and neither DCA nor cortisone added to the incubation fluid affects it significantly. Since injection of DCA or cortisone into normal mice likewise fails to affect the acid secretion of the stomach, "it is concluded that the hormones of the adrenal cortex play no direct or important part in the mechanism by which acid is secreted" [23]. Yet, cortisone appears to increase the resistance of the dog to the formation of the so-called "Mann-Williamson ulcers" [107].

The gastric ulcers produced by chronic *histamine* overdosage in the dog are allegedly also prevented by cortisone[12].

The vessels of the gastrointestinal tract are notoriously sensitive to the *periarteritis nodosa* producing action of M-C. Pronounced lesions of this type have been observed in rats overdosed with DCA[15].

Much further work will be necessary to elucidate the complex mechanism through which stress may cause gastrointestinal ulcers. Corticoids undoubtedly participate in this response, since adrenalectomy sensitizes animals to the production of gastric erosions by stress, while corticoid therapy largely counteracts this sensitization (cf. "Stress"). On the other hand, it is possible that excesses of G-Cs may likewise predispose the mucosa to erosion, as suggested particularly by the above mentioned clinical observations. Such a dual effect would not be without precedent, since both adrenalectomy and excess G-Cs also inhibit growth; yet, the growth-retardation caused by suprarenalectomy can be abolished by small doses of G-Cs

*Adrenergic hormones* also exert an important influence upon the function of the gastrointestinal tract. Nor-adrenalin is somewhat less effective than adrenalin in inhibiting the contractions of the intestines[140].

Intense muscular contraction, produced by farradic stimulation, blocks the inhibitory action of adrenalin upon the gastrointestinal tract of the dog[57]. The mechanism through which muscular activity exerts this effect has not yet been clarified.

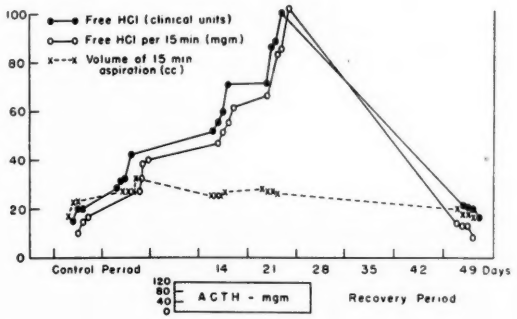


Fig. 5—*Effect of ACTH upon gastric acidity.* The administration of ACTH to normal subjects in doses of 100-160 mg. daily, over a period of 3-4 weeks, produced a marked increase in the secretion of gastric acidity (approximating 200 per cent) to the active duodenal ulcer level. Upon discontinuation of ACTH the gastric acidity returned to normal. (*Courtesy of Drs. S. J. Gray, J. A. Benson, Jr., R. W. Reifenstein and H. M. Spiro.*)

*Nervous system:*—The extensive literature concerning the effect of the vegetative nervous system upon the production of gastric and duodenal ulcers has been discussed elsewhere (cf. "Stress"). Let us add here that peptic ulcers may appear following thoracolumbar sympathectomy for hypertension in man[60, 111]. It remains to be seen whether this is due to predominance of vagal stimuli.

## Gastrointestinal Diseases of Adaptation

*Gastrointestinal ulcers:*—Several investigators have recently expressed the opinion that derailments of the G-A-S participate in the production of gastroduodenal ulcers in man[9, 41, 53, 75, 92]. The fact that various stressors can produce acute gastric erosions simultaneously with other manifestations of the A-R, has already been mentioned (cf. above). We must differentiate clearly, however, between the acute gastrointestinal erosions and hemorrhages caused by such alarming stimuli as burns, trauma, fulminating infections and intoxications, etc., on the one hand, and the chronic peptic ulcer on the other. We have no proof that the acute

gastric erosion of the A-R would tend to become chronic and transform itself into a peptic ulcer. There is much to suggest, on the other hand, that long lasting or oft repeated exposure to stress may participate in the causation of the latter.

Among the stressors likely to play a rôle in the pathogenesis of peptic ulcers in man, *nervous and emotional* stimuli are of special importance. Many clinical investigations suggest that lesions in the central nervous system (especially the hypothalamic region) and particularly psychic strain can cause hyperemia, increased secretion and motility finally resulting in chronic peptic ulcers in man[14, 33, 64, 76, 88, 130, 132, 133, 141, 143, 146].

In connection with the rôle of neurogenic stressors in the pathogenesis of peptic ulcers, it is perhaps also pertinent to mention the allegedly dramatic improvement that has been obtained in many cases by long-lasting treatment with repeated exhibition of *barbiturates*, in doses adequate to produce almost constant sleep for several days[19, 79]. This type of treatment may well act by elimination of neurogenic

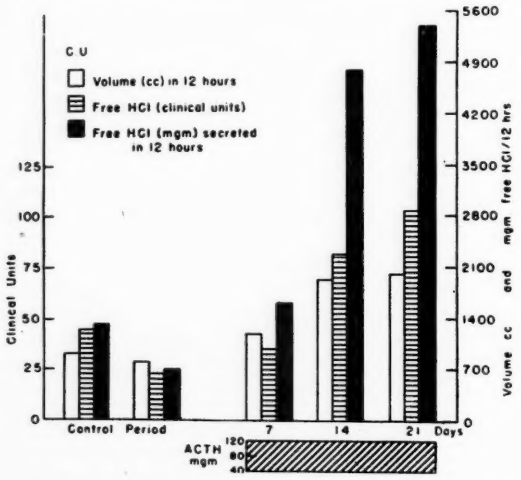


Fig. 6—*Effect of ACTH upon gastric acidity.* The administration of ACTH to a patient with a healed inactive duodenal ulcer produced a 12-hour nocturnal secretion of 5,550 mg. of free HCl. This level of acidity is significantly higher than that usually observed with active duodenal ulcer. (*Courtesy of Drs. S. J. Gray, J. A. Benson, Jr., R. W. Reifenstein and H. M. Spiro.*)

and psychogenic stress stimuli. Therapeutic *vagotomy* (cf. "Stress"), no doubt, also acts in this sense.

Ganglionic blocking agents such as TEA decrease the secretion and total acidity of the gastric juice in patients with gastrointestinal ulcers. This may be accompanied by radiologic evidence of improvement[80]. The effect is presumably due to the elimination of noxious nervous stimuli.

The 17-KS excretion is usually low during the active stage of peptic ulcer[59] but this may be an entirely nonspecific change, since it also occurs with many other diseases.

Treatment with large doses of ACTH and cortisone tends to aggravate the course of clinical peptic ulcers and it may, perhaps, even produce such lesions in previously healthy individuals[49, 62, 106, 107].

The administration of ACTH to a patient with gastric ulcer sometimes resulted in severe exacerbation of epigastric pain and tenderness, with definite evidence of peritoneal irritation and localized protective muscle spasm. At the same time, blood appeared in the stools and the secretion of *uropepsin, gastric pepsin and HCl* rose. Essentially similar changes have also been observed after adrenalin injection concurrently with the usual eosinopenia[44]. These observations may throw some light upon the mechanism through which ACTH could aggravate gastric ulcers.

Beneficial results have, allegedly, been obtained by small doses of *corticosterone* in one case of peptic ulcer[82], but this claim requires confirmation.

*Ulcerative colitis:*—We have an abundance of clinical data suggesting that *emotional and nervous* stimuli can cause important changes in the appearance and function of the colon and may participate in the development of ulcerative colitis in man[2, 30, 41, 43, 83, 101]. The gastrointestinal tract has often been called the sounding board of the emotions. Its clinical response to stress and "alarm" is well documented. It has been postulated that such diseases as peptic ulcer and ulcerative

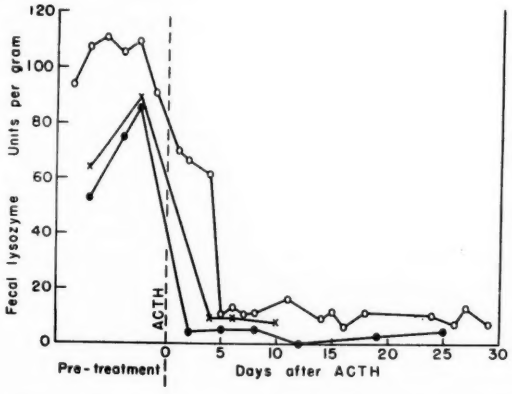


Fig. 7—*Effect of ACTH upon fecal lysozyme in ulcerative colitis.* ACTH was administered in doses of 100 to 160 mg. daily to three patients in the acute phase of ulcerative colitis. A prompt fall in fecal lysozyme titer occurred within the first five days of therapy accompanying the clinical remission of the disease. This response reflects an altered tissue reaction to injury. (*Courtesy of Drs. S. J. Gray, R. W. Reifenstein, J. A. Benson, Jr. and Young, J. C. Gordon.*)

colitis result from chronic anxiety and emotional stress reflecting an "alarm state"[46].

From our point of view, it is perhaps even more important to note that ulcerative colitis tends to be associated with *pyelonephritis or glomerulonephritis* in certain patients[67]. The occasional case of chronic ulcerative colitis with gynecomastia already suggested some relationship between this malady and the *endocrines*[74], but it is especially noteworthy that, judging by the response of the patient to adrenalin (eosinophils, 17-KS and corticoid excretion), impairment of "the reserve function of the adrenal cortex is not infrequently an accompaniment of chronic ulcerative colitis"[100]. All these data intimate, but do not prove, that a derangement of the G-A-S is of special importance in the pathogenesis of this disease.

In view of the close relationship between ulcerative colitis and the G-A-S, numerous experiments have been performed to examine the effect of "stress-

hormones" upon this disease. "The availability of such hormones as the adrenocorticotrophic hormone presents the opportunity of producing 'alarm' and studying its effect upon the gastrointestinal tract" [46]. In fact, it has been found that treatment with ACTH and *cortisone* frequently causes dramatic remission of the arthritis in those patients with ulcerative colitis who also suffer from joint involvement. At the same time, there is subjective improvement and often—though not always—definite proctoscopic or roentgenographic evidence of healing[25, 26, 36, 45, 46, 54, 56, 68, 70, 91, 93, 96, 104].

Attention should be called to the possibility that ACTH may precipitate bleeding or even perforation in certain cases of ulcerative colitis, somewhat as it can facilitate the development of a gastric ulcer. "In one center, for example, four consecutive examples of massive bleeding had occurred during treatment of chronic ulcerative colitis by ACTH, in one of which an emergency ileostomy was performed in the hope that diversion of the fecal stream would help in promoting hemostasis" [31]. (Although I have not seen such a case myself, I may add parenthetically that several physicians wrote me during the past few months to inquire whether such cases had been seen elsewhere, because they had observed such instances.)

The erythrocyte *cholinesterase*, which tends to be high in ulcerative colitis, falls under the influence of successful ACTH therapy parallel with the remission[93].

Since, recently, HPC (hydroxy-phenyl-cinchoninic acid) has been recommended for the treatment of rheumatoid arthritis, let us also mention the case of one patient, with chronic ulcerative colitis, in whom this drug apparently caused a striking amelioration of the disease[84].

The sometimes favorable therapeutic effect of *shock treatment*[22] may be mediated through an endogenous ACTH discharge, but, of course, it must also be kept in mind that this disease is often quite readily influenced by *psychotherapy*[13, 85] and may show spontaneous remissions.

*Nontropical sprue:*—Nontropical sprue (idiopathic steatorrhea) is frequently associated with morphologic and functional manifestations of hypocorticoidism and nonspecific stress has long been known to participate in the pathogenesis of this disease. In particular, various *infections* and *allergies* are believed to be capable of causing nontropical sprue[90] and metabolic studies suggested that this disease is associated with some degree of adrenocortical insufficiency[7, 34, 112]. Therefore, several papers published during the past year, attempted to interpret nontropical sprue as a *Disease of Adaptation*[16, 92a, 110].

As judged by the *adrenalin* eosinopenia, the ability of patients with typical sprue to discharge ACTH and G-Cs is essentially normal[97]. It is perhaps significant, however, that ACTH and *cortisone* treatment produced considerable improvement in several cases of nontropical sprue[3, 135] and, allegedly, even DCA may be of some value[7], hence, the possible rôle of the adrenal cortex in its pathogenesis deserves to be further explored.

*Appendicitis:*—Earlier data suggesting that appendicitis may be a Disease of Adaptation (cf. "Stress") have been supported by recent animal experiments. It was shown that a variety of stressors (e.g., formalin, histamine, anoxia) can produce appendicitis-like lesions in the rat, these always being accompanied by other typical manifestations of the A-R[35]. Conversely, it has been claimed that "diseases of Selye's General Adaptation Syndrome were more common amongst the appendectomized than amongst the controls" in a large series of children recently examined from this point of view[10].

In connection with the experimental production of appendicitis-like lesions with histamine, or with stressors capable of histamine liberation from tissues, it is

also noteworthy that the blood-histamine content is allegedly often augmented in patients with appendicitis and that anatomically the lesions are frequently reminiscent of *allergic* reactions[18]. Sometimes such allergic appendicular lesions may be associated with characteristic morphologic changes in the *nerve* endings[77, 78].

The probable participation of allergic and nervous reactions in the pathogenesis of appendicitis has recently been supported by extensive clinical and anatomic studies[61]. The frequent occurrence of the *periarteritic* lesions in the affected appendix[50] might also be taken as additional evidence indicating some relationship between the hyalinosis syndrome and this disease.

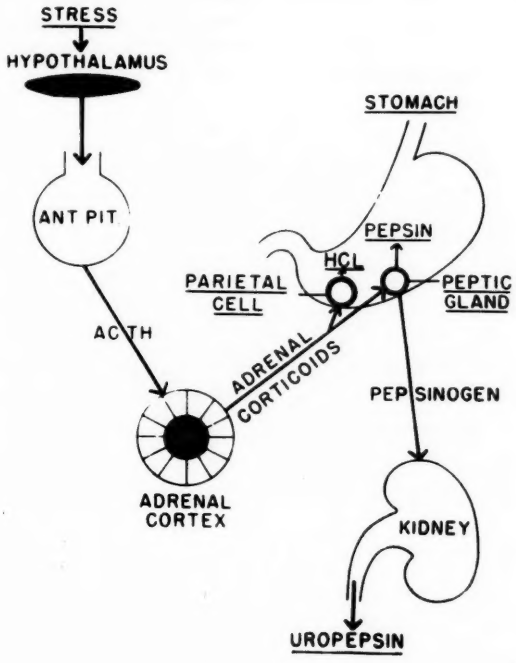


Fig. 8—*Hormonal pathway by which stress may be mediated to the stomach.* Stimulation by way of the hypothalamic-pituitary-adrenal axis with a resulting increase in gastric secretion of acid and pepsin and an elevation in the blood-pepsinogen and urinary uropepsin. (*Courtesy of Drs. S. J. Gray, J. A. Benson, Jr., R. W. Reifenstein and H. M. Spiro.*)

It is perhaps also significant that appendicitis may complicate the syndrome of "abdominal rheumatism" [105].

All these observations certainly do not suffice to prove that G-A-S derailments play an important part in the pathogenesis of appendicitis in man. Yet, evidence is accumulating to show that purely local damage to the appendix is not necessarily the most common evocative agent. Systemic neurohumoral reactions probably also play a rôle, perhaps by facilitating the acute disintegration of the lymphatic tissue and thus opening the way to enterogenic infections.

*Other gastrointestinal diseases:*—The various forms of the so-called *regional* and *necrotizing enteritides* are probably not manifestations of a specific infectious

disease, as had been previously thought, but may be due to an *allergic* reaction[21, 65, 73], *emotional* disturbances[134] or other systemic stressors. Significantly, such lesions have occasionally been found to occur in combination with a circumscribed hyperplasia of the *adrenal cortex*[37]. They allegedly begin with inflammatory *periarteritic* vascular lesions in the gut, which tend to undergo fibrinoid necrosis[6].

It is especially interesting that in a few cases of regional enteritis, ACTH proved to be of considerable value[45, 96].

In familial *polyposis* and in *pseudopolyposis* of the large bowel, ACTH treatment may cause marked regression of the vegetations[26] but the possible relationship of this disease to the G-A-S has not been examined.

The so-called *lipophagic intestinal granulomatosis,* (also known as *intestinal lipodystrophy* or Whipple's disease), is a chronic malady, characterized by the development of arthritis, cachexia, extreme asthenia, diarrhea, postprandial abdominal pain, an increase in the volume of the abdomen, dyspnea, cough and peripheral

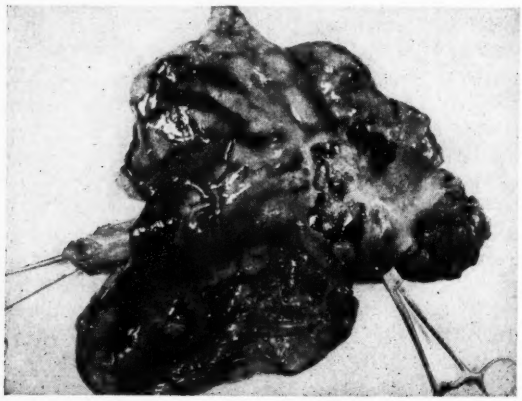

Fig. 9—*Gastric ulcers arising during ACTH treatment in man.* Perforated peptic ulcer at the lesser curvature of the stomach and a large ulcer at the cardia in a patient receiving 1,480 mg. of ACTH over a period of 19 days. The urinary uropepsin excretion was considerably elevated in this patient. There was no previous history of peptic ulcer. (*Courtesy of Drs. S. J. Gray, J. A. Benson, Jr., R. W. Reifenstein and H. M. Spiro.*)

edema. The mucosa of the small intestine often shows degenerative changes and contains many fat-laden macrophages, the so-called "lipophages". The disease is frequently accompanied by extensive granulomatosis, hyalinosis, fibrinous pericarditis and signs of hypocorticoidism. It appears to be elicited by a variety of nonspecific stressors[99] and allegedly "one cannot deny that the recent clinical and biologic studies concerning intestinal lipodystrophy bring a singularly convincing support to Selye's concept of the general-adaptation-syndrome. They suggest, furthermore, that treatment with cortisone and ACTH would be logical" [103]. However, such therapeutic attempts have not yet been published, at the time this book goes to press.

In patients with so-called *gastrointestinal allergy,* treatment with concentrated adrenocortical extract produced marked improvement, simultaneously with a sharp fall in blood-eosinophiles. This has been viewed as suggesting that here—as in many other allergic conditions—corticoid treatment acts by correcting a relative lack in G-Cs[102].

In a large series of newborn infants, *esophagitis* was an unusually common autopsy finding. "It is suggested that in many of the present cases esophagitis is a shock lesion. In the neonatal group of patients, shock may have been the result of various types of birth trauma, including asphyxia" [47].

Finally, it has been emphasized that in cases of bacillary *dysentery*, mild signs of Addisonism are commonly observed; these tend to be followed by a transitory phase of adrenal hyperfunction, during convalescence. Hence, it was concluded that adrenal malfunction plays an important part in determining the manifestations of this disease[58]. The claim that DCA exerts a favorable effect upon the course of dysentery[95] requires confirmation. On the other hand, we were able to cite a large number of clinical observations in support of the view that the G-A-S can modify the course of various systemic infections[126] and there is no reason to doubt that this also applies to gastrointestinal infections.

## Summary and Conclusions

In the preceding pages, we attempted to give a concise outline of the general-adaptation-syndrome and of those "diseases of adaptation", which are of special concern to the gastroenterologist.

Any effort to classify diseases will necessarily lead to some overlap between the individual groups which we delimit for this purpose. For instance, a case of scarlet fever may be listed among the infections (according to etiology) or among the renal and cardiac diseases (according to manifestations), if it causes myocarditis and nephritis. Similarly, depending upon our point of view, a case of typical Cushing's disease might be classified among the neoplasms (basophil adenoma of the anterior lobe), the derangements of carbohydrate metabolism (hyperglycemia, glycosuria), the skeletal diseases (osteoporosis), the endocrine disturbances of the pituitary (increased ACTH production), or the malfunction of the adrenal cortex (increased corticoid production).

This inherent weakness of any system of classification is tacitly understood by those who propose it. However, it is impossible to deal with diseases, without arranging them according to some principle. Hence, it has always been, and will always remain, an essential prerequisite for medicine as a science to classify the individual disease entities according to the system best suited to bring out their essential characteristics for further investigation.

A good deal of evidence has now accumulated in favor of the view that derangements of the G-A-S, especially anomalies in ACTH and corticoid production, play an important part in the pathogenesis of certain clinical syndromes, such as gastrointestinal ulcers, ulcerative colitis, etc.

It is hoped that this synopsis will stimulate proctologists to reinvestigate those disease entities which they currently encounter in their practice, in order to determine to what extent (if any) the G-A-S participates in their pathogenesis.

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# RECTOSIGMOIDAL POLYPS*

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## Introduction

One of the purposes of this paper is to present a resume of 25 years experience with the treatment of rectosigmoidal polyps. Another purpose is to present a classification and set down criteria and terms for the pathological diagnosis of polyps to help overcome the confusion of diagnoses which exists today. This paper also presents statistics on the frequency of polyps found in cancer detection clinics, in a proctologic specialty as well as in autopsy findings.

## Etiology and Incidence

Adenomatous polyps of the rectosigmoid are true tumors and their etiology is as yet unknown, as evidenced by the multiplicity of theories such as: allergy, hyperplasia of the mucosa, infection, irritation, heredity, and many others. The incidence of polyps varies according to the literature, and the discrepancy in these figures is apparently dependent upon the method of study, as well as the source of the material, i.e. whether in a general practice, a proctologic practice, cancer detection center, by x-ray or by autopsy figures. Nevertheless, a short review of some statistics should give ample evidence of the comparatively high incidence of polyps.

In Cancer Detection Centers the figures range from 1.6 to 8.8 per cent as noted:

| | |
|---|---|
| Ortmeyer of Women's and Children Hospital Cancer Prevention Clinic.[1] | 1.6% |
| Christianson of Cancer Detection Center, University of Minn.[2] | 4.6% |
| Yater Clinic, Washington, D. C.[3] | 8.8% |
| Goldman, Kolow, Altman, Rosenstein, Levy[4] | 2.3% |
| Kips Bay, Yorkville Cancer Detection Center[5] | 6.4% |
| Strang Cancer Prevention Clinics[5] | 6.4% |

Autopsy figures range from 3.37 to 21.40 per cent as indicated:

| | | | |
|---|---|---|---|
| Lawrence[6] | 7000 | autopsies | 3.37% |
| Sussman[7] | 1100 | " | 6.00% |
| Hellwig[8] | 1460 | " | 9.50% |
| Swinton and Haug[9] | 1843 | " | 7.00% |
| Klemperer[10] | | | 21.00% |
| Feyrter[11] | 1800 | " | 21.40% |

Figures in proctologic practices range from 1.2 to 7.49 per cent as indicated.

In the proctologic practice of two of us (M.G.S. and L.M.) the figures revealed 7.4 per cent over a period of 5 years.

| | |
|---|---|
| Martin[12] | 4.4% |
| Buie[13] | 2.5% |
| Thiele[14] | 1.2% |
| Brust[15] | 5.0% |
| Phillips[16] | 5.0% |
| Colbert[17] | 2.3% |
| Castro-Ault, Smith[18] | 2.4% |

* Read before the Third Annual Convention of the International Academy of Proctology, Atlantic City, N. J., June 7, 8, 1951.

Greer[19] ..... 3.0%
Spiesman and Malow ..... 7.4%

The location of polyps is of great importance. It is the consensus of opinion, and shown by statistics that the majority of rectosigmoidal polyps are found within the reach of the 10″ sigmoidoscope.

Males seem to be more frequently beset by polyps than females, 3 :2, and when one finds a rectal polyp, a diligent search should be made for others, inasmuch as multiple polyps are not infrequent.

Some statistics and the corresponding impressions of various surgeons and proctologists relative to the malignancy index of these polyps is very enlightening. Bacon and Broad[20] report 4 per cent malignancy found in their series of polyps: Christianson[2] reports 7.9 per cent; Castro,[18] Ault and Smith 15 per cent. Bacon[21] also reports that in the cases of proven rectosigmoidal malignancies, polyps were found in 31.2 per cent of these cases, and of these polyps, 6-14 per cent were malignant. Dukes[22] believes that all rectal polyps become malignant. Fitzgibbon[23] believes that all cancers of the bowel arise from polyps. Sawyer[24] contends that all

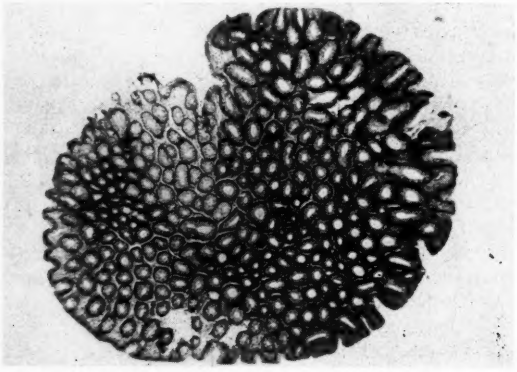

Fig. 1—Section of benign resting polyp revealing small round glands which are quite regular in size and shape.

polyps of the rectum are potentially malignant and that the probability of carcinoma increases with the number of polyps present. In support of this last statement, is the almost certain development of malignancy in cases of polypoidosis of the colon.

Biopsies are routinely taken on all polyps except those under 5 mm., but should not be done unless the necessary equipment is on hand to control hemorrhage or aspirate smoke and blood from the area of bleeding. Hemorrhage from these polyps, following biopsy, can be quite formidable unless the operator is properly prepared.

A point that should need no repetition but deserves it, is that those patients complaining of rectal bleeding and having internal hemorrhoids, should be sigmoidoscoped before any hemorrhoidectomy is performed. Also if a solitary polyp is present, a search for other growths above this polyp is essential.

It is our practice to order barium enema study on all patients having one polyp, to rule out the possibility of more colon polyps up higher. This examination, as performed in many laboratories or hospitals will fail to reveal colonic polyps, especially in the sigmoid, hepatic and splenic flexures, unless a contrast barium study, with lateral and oblique views of the sigmoid, splenic and hepatic flexures are included. These extralateral and oblique views reveal pathology often missed by

the routine colon examination. Fictitious polyps are frequently seen on barium enema and a suggestion as to diminishing the frequency of these misleading findings is found in an article which suggests the following:

1. No supper the previous day.
2. 6 P.M. previous day—take 1 oz. castor oil.
3. Begin at 6 A.M., take 3 soapsuds enemas at 15 min. intervals.
   (Soap on enema tip)
4. Small breakfast at 7 A.M.
5. X-ray at 8 A.M.

Routine proctosigmoidoscopy is urgently recommended for all persons over 45 years of age because of the increased incidence of colon polyps above that age. The following figures of polyps relative to age is from the Strang Cancer Prevention Clinic.[5]

TABLE I

| *Age (Years)* | *Number of Polyps* | *Per Cent* |
|---|---|---|
| 13 | 1 | 0.2 |
| 20 to 29 | 4 | 0.8 |
| 30 to 39 | 47 | 9 |
| 40 to 49 | 177 | 37 |
| 50 to 59 | 178 | 38 |
| 60 to 69 | 61 | 12 |
| 70 to 79 | 10 | 2 |
| 80 | 1 | 0.2 |

## Pathology

There is much disagreement as to the proper classification of rectal polyps. This is best exemplified by the experience of Marie Ortmeyer of Chicago, who sent 19 biopsied polyps to 3 pathologists for diagnosis. All 3 agreed on the diagnosis of only 3 of these biopsies. The differences of opinion were probably due only to variation in terminology and differences in the criterion of malignancy. Thus, what one pathologist will call carcinoma *in situ*, preinvasive, another will call suspicious for carcinoma; another will call benign polyp with atypical changes in the mucosa, and still another will call proliferating adenoma. There is usually no disagreement with the benign polyps or with infiltrating carcinomatous polyps. This controversy over the diagnosis of carcinoma *in situ* has arisen in many other regions, such as, the urinary bladder, cervix, skin, etc. The criteria for the diagnosis of carcinoma *in situ* of the cervix, skin, and bladder polyps are similar and they have been almost universally accepted. However, there seems to be a tendency among pathologists to designate carcinoma *in situ* of rectal polyps by other terms, such as, suspicious for cancer, polyp with atypical cells and rapidly proliferating polyps.

The *benign resting polyp* contains normal epithelium (Fig. 1) consisting of columnar cells with light staining cytoplasm and small nuclei located in one row at the base of the cells. Some of the cells are goblet type because they are bulging with mucus (Fig. 2). On cross section these polyps reveal round and regular glands. In some rectal polyps that appear to be actively growing we see a change in the architecture of the polyp. The glands become larger and somewhat tortuous (Fig. 3). The glands frequently contain basophilic staining mucus. However, the cells contain pale cytoplasm and the nuclei are in one row at the base of the cell and assume a higher position in the cell (Fig. 4). This change is still benign and we call it an *actively proliferating polyp*. Occasionally we see this picture associated with the next group, carcinoma *in situ*. The definite carcinomatous change, intra-

epithelial, occurs when there is a piling up of nuclei and cells in two or three rows, the nuclei are definitely larger and more pleomorphic and the cytoplasm is more violet. Usually the transformation from normal to carcinomatous epithelium occurs without intermediate phases; the dislocation of the nuclei may be preceded only by the darker staining of the protoplasm (Fig. 5). Frequently, normal glands are

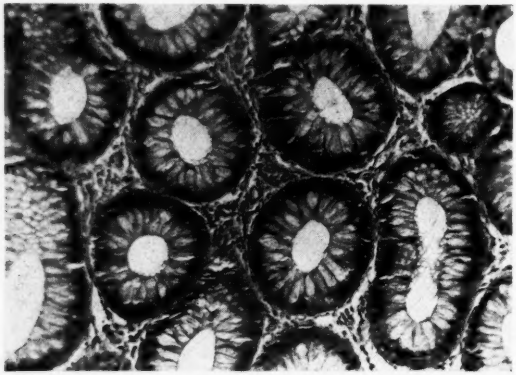

Fig. 2—High power magnification of benign resting polyp revealing normal rectal epithelium consisting of columnar cells with light staining cytoplasm, small nuclei located in one row at the base of the cells and occasional goblet cells.

seen next to carcinomatous glands. Sometimes the lining of one gland consists of normal and of carcinomatous epithelium separated by a sharp borderline with no transitional cells between the last normal and the first carcinomatous cell. We believe, as Dr. Schiller does, that these are the earliest carcinomatous changes, and that this group should be classified as *carcinoma in situ* (Figs. 6, 7).

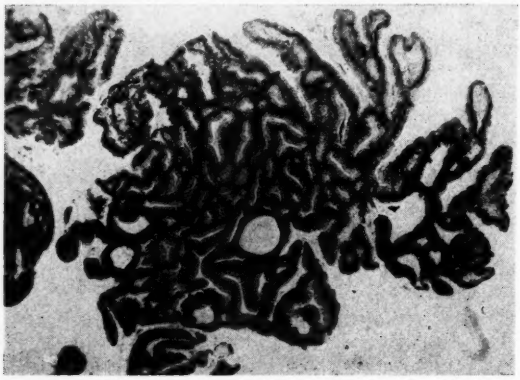

Fig. 3—Section of actively proliferating benign polyp showing larger glands which are tortuous and irregular.

It has been our experience that early malignant changes in a polyp occur more often at the periphery than at or near its base. The polyp with infiltrating adenocarcinoma will show changes at the periphery and at the base, infiltrating into the muscularis mucosa. The infiltrating of these malignant glands into the bowel wall at the base of the polyp is the criterion for #3 in our classification, i.e. *polyp with*

***infiltrating adenocarcinoma*** (Fig. 8). Thus, our suggested classification of rectal polyps is as follows:

1. *Benign Polyps*
   a. Resting Polyps
   b. Actively Proliferating Polyps
2. *Polyp With Carcinoma In Situ*
3. *Polyp With Infiltrating Adenocarcinoma*

That all polyps are potentially malignant is well borne out in current literature. It is not infrequent to see numerous polyps in association with frank adenocarcinoma of colon or rectum. A study of these polyps usually shows the entire pathogenesis of large bowel carcinoma. Some of the polyps will be benign, resting type or actively proliferating, while some will show carcinoma *in situ* and some will show infiltrating adenocarcinoma in the base of the polyp. It is for this reason that multiple carcinomas of the rectum and colon are not infrequent. Intraepithelial carcinomatous changes in a polyp may be confined to one portion of a polyp. Frequently, punch biopsy of a polyp may not include a malignant portion and it is

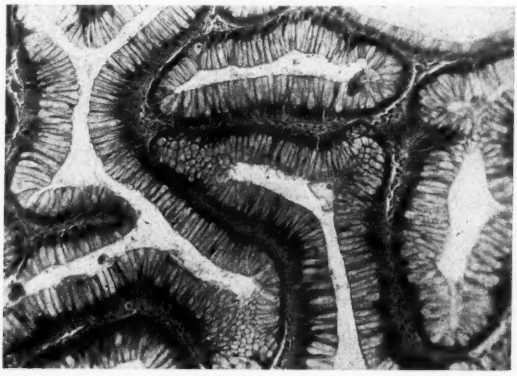

Fig. 4—High power magnification of actively proliferating polyp revealing the tortuosity of the glands, with the cells longer, thinner and piled up. The nuclei, however, are still in one row at the base of the cells indicating benignancy. This group has been called "premalignant", "pseudomalignant", "suspicious for carcinoma", proliferating adenoma or benign polyp with atypical changes".

for this reason that a simple punch biopsy may be misleading and why the entire polyp should be removed for microscopic study.

## Symptoms

Most patients with rectosigmoidal polyps have no complaints indicative of their presence, although bleeding is the most common complaint, occurring in 50 per cent (according to Swinton and Warren[27]) and 8-50 per cent (Strang Clinic[5]). In a series of 391 cases reported by Bacon and Broad[28], 63 per cent offered no complaints referable to their growths. Protrusion of a mass through the anus is sometimes noted, as is also a complaint of a feeling of fullness or pressure in the rectum. In our experience rectal bleeding caused by polyps occurs only occasionally. The greatest percentage of polyps was found on routine sigmoidoscopy.

## Diagnosis

The diagnosis of rectal polyps is based primarily upon sigmoidoscopic examinations although digital examination and x-ray study should not be omitted. As evi-

denced by the statistics quoted and the paucity of symptoms in these cases, it becomes mandatory for every physician to look for these growths by routine sigmoidoscopy. It is a relatively safe and simple procedure but only too often, omitted from a general examination.

### Treatment of Rectosigmoidal Polyps

The treatment of polyps is usually not difficult, but in questionable cases, is best determined by one having had years of experience with this type of work. To avoid confusion, the writer will refer to the types of high frequency currents commonly used for the removal of polyps, in the following manner. The word fulguration, which refers to either the single or the double pole type of currents will not be used hereafter, instead, when discussing the single pole sparking current, we will refer to desiccation, and when discussing the two pole current, we will refer to coagulation.

The biopsy punch, followed by desiccation is the instrument and current most commonly used. For low lying accessible lesions, excision and suture is preferred; while lesions beyond the reach of the sigmoidoscope require removal through a colostomy incision. If the polyp has invaded the wall of the colon a resection of a

Fig. 5—Polyp with carcinoma *in situ*. Note the darker staining cytoplasm, the piling up of nuclei in two or more rows (arrow), and the larger and more pleophormic nuclei. This group has also been called "suspicious for cancer" and "polyp with atypical changes".

portion of the colon is indicated. Multiple polyps are treated the same as solitary ones, by the biopsy punch and desiccation method; while the familial type of polypoidosis which cover the entire rectum and colon should have only one form of therapy, namely, colectomy with ileorectostomy, followed later by desiccation of the rectal polyps.

For the inexperienced operator the desiccation current for small sessile polyps is the safest, because the current does not penetrate deeply. In the hands of one experienced, and with a good working knowledge of the high frequency machine and depth of penetration of the ball tip being used, the two pole diathermy current is satisfactory for lesions below the peritoneum.

### Sessile Polyps Above The Peritoneal Reflection

For sessile polyps less than 1 cm. in diameter above the peritoneal reflection, our usual routine is to remove most of the polyp with the biopsy punch and desiccate the base and surrounding mucosa. One should have electrodes with suction attachments capable of removing smoke to promote better visibility and avoid small gas explosions and also capable of removing blood in case of extensive hemorrhage.

For sessile polyps more than 1 cm. in diameter above the peritoneal reflection, carcinoma with invasion should be suspected, especially if the least degree of firmness exists. The problem becomes a precarious one requiring extensive judgment and experience. Because it is difficult to reach and examine the polyp, one has to depend upon vision, sigmoidoscopic palpation, biopsy and the "Pap" smear to arrive at a proper decision. If fixation and induration is felt when the end of the sigmoidoscope is pressed against an ulcerated large bleeding polyp and the Papanicoalau stain and biopsy specimens show malignant degeneration, the patient should have an exploration and resection of a portion of the colon. Careful search should be made for metastatic glands, and a thorough search for more existing polyps.

### Pedunculated Polyps Above The Peritoneal Reflection

Although many men describe the snare method of coagulation for polyps in proctologic papers and books, personal conversation with many of these men, reveals the fact which we have always shared, that the snare method is fraught with danger. Pedicles of polyps frequently carry large arteries. Unintentional undue traction may tear off a pedicle resulting in excessive hemorrhage at a point which

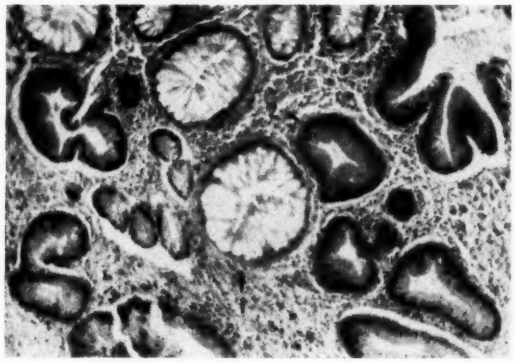

Fig. 6—Polyp with carcinoma *in situ*. Note normal glands (1) alongside malignant glands (2) in high power magnification.

is very difficult to expose and reach. Excessive coagulation to control hemorrhage may result in slough and perforation of the sigmoid colon.

Because of traction by the polyp on the sigmoid wall over a long period of time, the pedicle may be a part of the sigmoid wall. It is quite obvious that removing such a pedicle with a snare would result in a perforation of the bowel wall with ensuing peritonitis. Even omentum and small intestine have been reported in the pedicles of pedunculated polyps. Using the snare method in such cases may be disastrous.

Instead of the snare we have found that the Frankfeld double cup electrode, using the diathermy (two pole) current and removing the polyp piecemeal by the fractional method is the safest for the greatest number of men. A portion of the polyp is grasped by the cups and coagulated, one or more pieces being done at one sitting. This can be repeated two or three times a week. When only the pedicle remains, care must be taken not to coagulate too deeply at the base of the pedicle to avoid slough and perforation. Too deep a treatment of the base of the pedicle may also result in a long-lasting punched-out ulcer. When the pedicle base is reached it is safer to use the desiccating current (1 pole) and repeat if necessary, to avoid the above possible complications. Again it must be repeated, that one must have

available, gas and blood suction equipment on hand at all times to remove explosable gases in the sigmoid, to improve visibility and to control possible hemorrhage. If hemorrhage should occur, a good combination suction and desiccation (1 pole) electrode could clear the field of blood and smoke, thus exposing the bleeding vessel and then seal it with sparking desiccation.

### Sessile Polyps Below the Peritoneal Reflection

Sessile polyps less than 1 cm. in diameter below the peritoneal reflection can easily be removed with a biopsy punch followed by desiccation of the base and surrounding mucosa.

Polyps larger than 1 cm. in diameter which are not ulcerated, do not bleed easily, are not indurated and fixed and are accessible to the operator should be excised in toto with a collar of basal mucosa. The exposed wound can then be closed with a suture.

Polyps more than 1 cm. in diameter below the peritoneal reflection not accessible to excision and showing ulceration, free bleeding, fixation, induration and positive Papanicoalau stain and biopsy findings are the real problem cases. To remove by

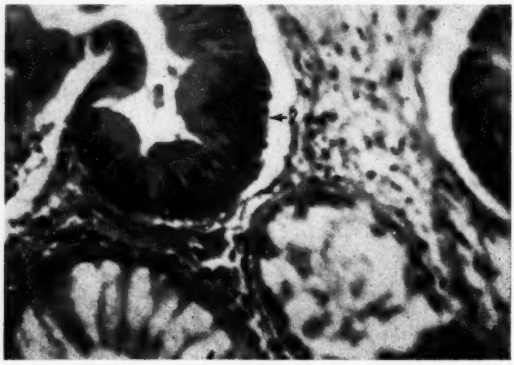

Fig. 7—Polyp with carcinoma *in situ*. Note normal glands (1) alongside malignant glands (2) in high power magnification.

fractional coagulation or to do a complete abdominoperineal resection becomes the serious question to be decided. These are the cases that tax the conscience of the proctologist or surgeon. We have always tried to place ourselves in the position of the patient and our decisions have always been based on what we would want done for ourselves if we had a similar lesion. The following is our usual conclusion in such cases. If a polyp with the above findings is not too large and is located on the posterior wall below the peritoneal reflection, we coagulate all of the lesion as well as a good size collar around the base. This type of case should be observed once monthly for years, as a recurrence may require radical surgery. If local recurrence does occur and if no invasion is found on pathological examination, further desiccation or coagulation may still be indicated, but is associated with heavy responsibility. The experience and judgment of the proctologist will have to determine the course of treatment in these cases. If local recurrence shows invasion, then radical resection is the treatment of choice.

For malignant types of large polyps on the anterior wall of the rectum, the decision for treatment is associated with an even greater responsibility because of the close proximity of the bladder, uterus, seminal vesicles and prostate gland. Here

again, one's personal experience with this type of lesion will determine the type of treatment to institute.

Since villous tumors are rarely found above the peritoneal reflection, we have had good results with excision en masse. Since a certain percentage of these tumors are malignant at their base, the postoperative periodic observations are most important. Frequently, small residual areas have to be desiccated or coagulated. In our experience, excision and suturing of the base has given us consistently good results. In one case we found an adenocarcinoma of the rectosigmoid about 4 cm. above an excised villous tumor. This patient was followed up by an abdomino-perineal resection.

### Pedunculated Polyps Below The Peritoneal Reflection

If pedunculated polyps below the peritoneal reflection are accessible to the operator, they are best removed by excision with an accompanying collar of rectal mucosa. The wound is sutured by the purse string method. If not easily reached, then the Frankfeld cup fractional method described above should be instituted.

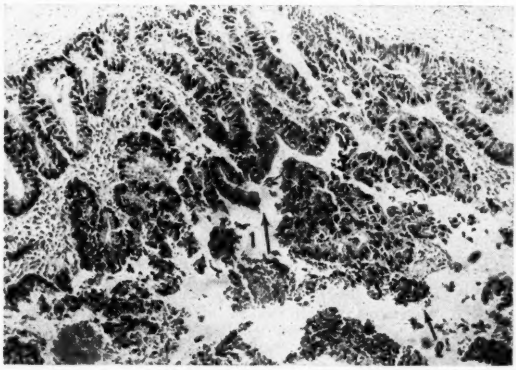

Fig. 8—Polyp with infiltrating adenocarcinoma. Glandular elements can be made out as well as broken down glands (1) and invasion by carcinoma cells (2).

Since some pedunculated polyps present areas of malignant change the question arises: should one remove the entire rectum in such cases or use coagulation locally. Our experience over a period of twenty-five years has taught us that when palpation reveals a freely movable, soft, nonindurated pedicled polyp and histologic studies disclose malignant change confined to a single area not involving the stalk or base, local excision, desiccation or coagulation is entirely satisfactory. Rankin and Johnson[29] have recently reported similar experiences. We have called these polyps histologically malignant and clinically benign. We must keep in mind, however, the possibility of a malignant change in a polyp being associated with established metastasis in regional lymph nodes.

### Considerations Following Polyp Therapy

One should be on the lookout for perforation or hemorrhage 5-10 days following the use of coagulation. Patients should not be permitted to work or to lift heavy objects for at least two weeks after such treatment.

In questionable polyp cases, 8-10 weeks should be allowed to transpire following coagulation before one decides about the results accomplished. If, after such a period the area appears healed, becomes mobile and pliable and adopts the appear-

ance of the adjacent tissue, the result will usually be satisfactory. However, if after such a period, the area is indurated, fixed, friable and the biopsy shows malignant tissue, radical surgery should be instituted; besides, further desiccation or coagulation produces perirectal inflammation and adhesions to the vaginal wall and other pelvic structures making radical surgery very difficult.

Any time after six weeks any suspicious tissue should be biopsied and the case again reviewed for further consideration.

## Summary

The statistical evidence relative to the incidence of rectosigmoidal polyps and their malignancy index is sufficiently high to warrant a constant search for their presence.

A classification of rectal polyps is offered to simplify and coordinate the various differences in terminology and criteria of malignancy and to eliminate the confusion that exists at present.

The treatment of rectosigmoidal polyps is discussed at length, the therapeutic approach depending upon the location of the polyp relative to the peritoneal reflection and whether it be sessile or pedunculated.

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# PERIANAL APOCRINE GLAND FISTULA*

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There frequently exists in the perianal, or more properly, the perineal, region a disease entity which is of equal interest to the proctologist and the dermatologist. It is characterized by abscess formation, draining sinuses, scarring and the ultimate formation of subcutaneous fistulous tracts. This pathological condition is interesting to the surgeon because its perianal manifestations are frequently confused with those of anal fistula, pilonidal sinus disease and other conditions which result in the production of chronic sinuses in this area. The dermatologist is aware that disease of the apocrine glands is all too prevalent in the axilla, the perineum and less frequently beneath the female breasts. Technically inflammation existing in the apocrine sweat glands, or sudoriferous glands, is known as *hidradenitis suppurativa.*

The apocrine sweat glands, by far fewer in number than the eccrine sweat glands, are prominently located in the posterior cervical region, axilla, under the breasts, in the pubic area, the vulva, and the perineal skin. They differ from ordinary sweat glands, which appear in fetal life, by their late occurrence at puberty as anlages of hair. Arey places them in areas where coarse hair is found. They differ also from the eccrine sweat glands in that they are large compound tubular glands which evacuate their cellular content through ducts into the hair follicle or onto the epidermis adjacent to the glands. The tops of their secretory cells break away along with the secretions. This is thicker than that of the eccrine glands and has a decidedly musty odor. Because they begin to function at, or after, puberty they are assumed to be sex glands, the vestigial remnants of scent glands.

The etiology of infection of the apocrine glands is actually unknown. Jackman believes the cause, although obscure, is infectious, and is associated with a variety of bacteria which reach the glands by several routes. Sutton and Marks ascribe it to dysfunction of lipid metabolism and decrease in thyroid function. Brunsting assures us that the disease is not tubercular in origin. Richter suggests a cholesterol imbalance, others have mentioned a granulomatous basis for the pathology. Equally as varied are the synonyms for this disease process; abscess or furunculosis of the buttocks, pyoderma of the perianal region, chronic undermining, burrowing ulcer of the skin, nonspecific granuloma and fistulous disease of the buttocks.

Velpeau described superficial abscesses involving the skin of the axillary, mammary and perineal regions as early as 1839, but he did not associate the disease with the sweat glands. Between 1854 and 1865, Verneuil, the French surgeon, made a series of publications and suggested the association of these abscesses with the sweat glands. Pollitizer in 1892 also allayed perineal abscesses to the apocrine sweat glands.

Perianal apocrine fistula disease is most common in robust individuals who have, or have had, acne, and are in the second or third decades of life. Older individuals will, on careful questioning, relate the onset of the disease to have begun during the latter period. A cycle of abscess formation, draining sinuses and ultimate extensive scarring is the general rule. One case in our series presented the chronic signs of widespread keloid-like scarring with scattered dimples, in the center of which was located a draining sinus opening. This thickening and elevation of the skin was present on each buttock, and ran from the sacrum to the scrotal skin.

* Read before the Third Annual Convention of the International Academy of Proctology, Atlantic City, N. J., June 7, 8, 1951.

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Brunsting describes the cellular reaction as originating in the subcutis, primarily within the lumen of the apocrine glands, and in the neighboring periglandular connective tissue. Clinically the early lesion appears as a solitary abscess, simulating closely a suruncle. As the infectious process continues, suppuration, burrowing, undermining and ultimate sinus tract formation takes place. The condition may regress and lie dormant for a long period of time, only to exacerbate and undergo the above chain of events. In time the subcutaneous tissue is honeycombed with the abscess cavities and sinuses. The sinus orifices vary slightly in size, ranging from pinpoint to 0.8 cm. in diameter. The larger, rounded openings may have a collar of elevated granulation tissue about them, much like the tissue seen in the later stages of healing of an operated pilonidal cyst where some irritating drainage is present. In a very recent case there were five scattered lesions on the left buttock measuring 0.5 to 0.6 cm. in diameter, with a flattened center covered by a yellow-gray purulent membrane, and surrounded by such an elevated collar of granulation tissue.

Histologically, Jackman describes the primary involvement as an inflammatory reaction. He states that infection spreads by means of the lymphatic ducts and the tissue spaces, to include the adjacent skin and subcutaneous tissue. Eventually, the glands are destroyed, and fail to regenerate. Polymorphonuclear leucocytes make an early appearance, followed by lymphocytes, and the suppurative stage is reached. If healing occurs at this stage a stellate scar is produced with a small centrally placed, occluded orifice. Of the advanced cases, Brunsting has microscopically demonstrated large pale-staining irregularly shaped, giant cells with deep-staining eccentrically placed nuclei. These late, or chronic cases, are characterized by fibrosis, especially prominent in the lower portions of the sections, and separating the diseased tissue from the healthy underlying adipose tissue. The upper cutis and epidermis are not involved until extensive destruction has occurred throughout the subcutis.

Color changes varying from light purple to light brown were observed in our cases, with the skin overlying the lesions presenting an atrophic appearance. The drainage, though purulent, is not great in amount, is not thick, and is distinctive in having a musty odor, in contrast to the drainage from true anal fistula which reminds one of hydrogen sulfide gas. In three of our cases we attempted to culture the discharge, but no true conclusion may be drawn. Staphylococcus aureus and citrus occurred in all three, while one showed streptococcus viridans.

The true pathologic value in knowledge of this disease is that in unusual instances, the burrowing process may enter the anal canal, infrequently traversing beneath the transitional skin, although always limiting itself before the dentate line is reached. Kratzer recently presented an excellent paper, enlarging the literature on the role of the anal ducts in the formation and origin of true anal fistula. Yet his study was concerned with twenty cases of pararectal abscess of whom he states, "They are similar to the classic group in that the abscesses occur in the usual locations. They are dissimilar in that an obvious primary or internal opening was not demonstrated." In our opinion these cases may have represented perianal instances of *hidradenitis suppurativa*, although the basis for Kratzer's surgery is obscure. Earlier we admitted that limitation of apocrine gland disease to the perianal region is shortsighted. A case studied substantiates this point, for several episodes of surgery were necessary to evoke a cure of the tracts which lattice-worked this man's scrotum and perineum. In this case the inguinal lymph nodes were palpable bilaterally. Geber was misled in his clinical observations, to which Brunsting prescribes, for he concluded that the disease about the anus

may exhibit extension through the deep tissues and may continue with eventual perforation of the rectum and the formation of anal fistulas.

It is not remarkable that too few cases of *hidradenitis suppurativa* about the anus have been recorded in the literature, for the first reference to the disease, as found anywhere in the body, was made in the English language in 1933 by Lane. Between 1936 and 1942 Jackman reported eleven cases. Brunsting wrote of twenty-two cases of *hidradenitis,* of which twelve involved the perianal region. In 1938 Smith described six cases in great detail. Of these, five had active acne, or scars resulting from acne, two had lues, and one had a sister with the disease. Making the point that coexisting anal pathology may often be present, Smith asserts that three of his cases had small anal fistulae, but these were inactive. We had one case make its appearance after a fistulectomy, and a second followed a hemorrhoidectomy—both in females. Smith's report consisted of five males and one female, while we have studied four males and two females. Christensen submitted case reports on six males and two females. Bacon's series consisted of five cases.

The onset of the disease is insidious. Early symptoms may be pruritus, burning or local tenderness. One or several slightly tender, indurated nodules will be discernible on inspection. Palpation may give a variable degree of firmness, and usually moderate tenderness. The nodule may undergo involution, though most commonly discharge occurs in about one week. The drainage is thin, mucinous and purulent, and most often gives a musty odor. If the lesions are not the first encountered by the patient, signs of old exacerbations are present in the form of stellate scarring surrounding a centrally located pore. The newer nodules most frequently occur adjacent to an original lesion. A small pustule may form at the summit of the nodule, although as a rule this has spontaneously drained by the time the patient is seen, leaving a lesion about half a centimeter in diameter, round in shape, covered by a thin purulent membrane, and often surrounded by a collar of heaped up granulation tissue. When the latter is lifted free, pressure on the adjacent parts causes the discharge of a few drops of pus from the centrally located pore.

In long standing cases, the sinus tracts can be palpated beneath the skin, and if they are inflamed they will be tender to touch. A probe passed through a tract will egress from the pore of each involved gland. As the infection commonly follows the natural skin folds, infections close to the anal verge will permit the introduction of the probe cephalid, but will either present the tip before the dentate line, or the probe will meet obstruction under the transitional skin. We have had examples of inguinal lymph gland enlargement, but only when the scrotal skin was involved.

There are few systemic signs. The temperature is not elevated. A slight leucocytosis with a moderate increase in lymphocytes was met with in some of our cases. In four of the patients studied we had a sedimentation rate done, and found it minimally elevated in all. Brunsting claims that gangrene doesn't occur, and nowhere else have we met with mention of this complication. We did not perform cholesterol, nor cholesterol ester determinations in our cases, although reference is made in the literature to specific changes in each.

Therapy shows variance in the literature. Sutton and Marks claim prompt and satisfactory results with a low fat diet and the administration of thyroid. Smith's cases, before subjection to surgery, were previously treated, without success, with various ointments, bacteriophage, ultraviolet light, local heat, vaccines, and x-ray. Jackman recommends incision and drainage of the individual abscesses followed by x-ray therapy in the early cases, and complete surgical

extirpation with, or without plastic repair in the chronic cases. Chemotherapeutic agents offer variable results when used alone, or with local measures such as sitz baths, or local heat. Christensen believes that large doses of penicillin will reduce and thicken the discharge, and bring quiescence to the acute inflammation, but will not heal multiple sinuses. Sutton warns against the use of ointments.

We have treated two early cases with incision and drainage, supplemented by sitz baths, large doses of penicillin and oral sulfadiazine. An apparent cure was evoked in both instances. Our older cases were dealt with surgically. A probe was inserted into each tract and the overlying skin incised using the electric knife. An attempt was made not to angulate the incisions for fear of retraction of the skin edges with production of a wide scar. Healthy tissue, whenever possible, was left between parallel incisions. When the tracts presented an acute angulation the surgery was carried out in stages. A back cut was utilized as a general rule. Care was exercised to cut down on the probe and not to carry the incision into the healthy underlying adipose tissue, as indicated by the presence of puffs of grayish smoke produced by burning fat. The base of each tract was curetted carefully, and a small amount of Escitol was brushed over the area. Packing was strictly avoided.

Postoperatively, penicillin was used for three to four days. Wet dressing, moistened with aqueous chlorophyll solution were immediately applied, with sitz baths instituted on the first postoperative day. Trichloracetic acid was employed to keep down exuberant granulation tissue. Cholophyll ointment containing 20 per cent benzocaine and oxyquinoline benzoate 0.4 per cent was frequently applied to promote healing and render the situation as painless as possible. No attempt was made to alter bowel habits, nor to change the personal preference as to diet. Healing was usually complete, or well along, by the fourth week. Only an average of nine days was lost from gainful employment.

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# SURGICAL LESIONS OF THE RIGHT HALF OF THE COLON AND THEIR TREATMENT*†

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Early diagnosis in lesions of the right half of the colon is fraught with difficulty. This may be due to the paucity of symptoms or attributing them to contiguous organs, such as the appendix or gallbladder, etc.

Carcinoma is the most frequent lesion (ten to sixteen per cent of all neoplasms of the colon region occurring in this area) and consequently has to be considered in all surgical conditions of this part of the colon, even in those presenting acute symptoms.

Acute volvulus of the cecum, with or without the ascending colon, occurs in people who have hypermotility of this portion of the bowel. In fact, in three per cent of all adults, the ascending colon has a mesentery, and twenty per cent of the population have marked hypermobility of the cecum. The symptoms are those of intestinal obstruction but the patient may be able to pass gas. The cecum of the ascending colon and even the hepatic flexure, if adequate fixation has not taken place, may twist on the longitudinal axis or it may rotate on the mesenteric axis. The latter is worse because of the danger of vascular embarrassment. If the cecum alone is involved, the rotation is apt to be counter clock-wise; if the whole right half of the colon is involved, the rotation is usually clock-wise. X-ray shows a large loop of distended intestine with clefts of colic character and an emptiness of the right iliac fossa which is more or less characteristic.

The treatment is reduction of the torsion with fixation of the cecum or cecostomy. The latter may be done particularly in the late cases.

A radical resection of the right half of the colon with transverse ileocolostomy or a Lahey-Mikulicz resection may be done if the gangrene of the bowel is extensive.

In recurrent volvulus, Dixon has advised raising a flap of peritoneum on the lateral aspect of the mesentery, bringing it over the cecum and sewing it to the mesial aspect of the mesentery, thus fixing the bowel permanently.

Diverticulitis of the cecum is often mistaken for appendicitis because of the abdominal pain, tenderness, rigidity and rebound tenderness, associated with rise in temperature, pulse and white blood count. The true diagnosis (unless previously x-rayed) is not discovered until the patient is operated upon. Sometimes the process is so severe that there is widespread involvement of the cecum and this, plus enlarged nodes in the mesentery, may give the impression of malignancy. If one side of the cecum is free, by palpating through to the opposite wall of the cecum, one may feel the mouth of the diverticulum.

* Read before the Third Annual Convention of the International Academy of Proctology, Atlantic City, N. J., June 7, 8, 1951.
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If the diverticulum is obvious and has a small neck, the base can be ligated and the diverticulum excised, or, if the base is broad, one may have to excise a portion of the cecal wall with it. With more widespread involvement one can exteriorize it and if the suspicion of malignancy is very great, a resection may be done in one or multiple stages.

Torsion or infarction of the appendices epiploices may occur. It usually occurs in well nourished, obese individuals, frequently in the sigmoid or the cecum. The patients are not too ill, and have a slight rise in temperature and pulse. Sharp or cramp-like abdominal pains with tenderness, rebound tenderness, with or without rigidity, is present. Excision of the affected appendage can be done, being careful to identify the presence or absence of a diverticulum often laden with

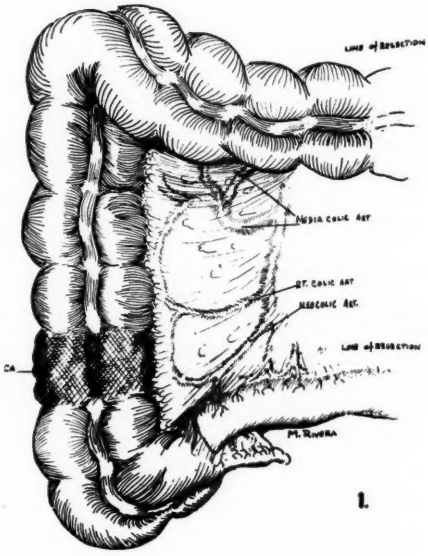


Fig. 1—Carcinoma of the ascending colon.

feces, which may be under the inflamed or gangrenous appendage. If the condition subsides without operation, fibrosis and even calcification may take place. The fibrosed appendage may form a band which may stretch across the colon, cause obstructive symptoms and even x-ray may give rise to doubt as to whether or not malignancy is present.

Intussusception may involve the right half of the colon if it is of the ileocecal, ileocolic or colocolic type. It usually occurs in children and is characterized by severe paroxysmal pain associated with pallor, the formation of a sausage-shaped tumor and the passage of blood and mucus by rectum. Shock may or may not be present.

X-ray, both flat plate and barium enema, often gives valuable information. Laparotomy with reduction of the intussusception is the procedure of choice. Resection and primary anastomosis may have to be done. Very young children withstand the Mikulicz procedure very badly. Intussusception is often associated

with a tumor in the lumen of the bowel and in older people it may be the site of chronic obstruction. X-ray will usually differentiate it from malignancy, however, some times, particularly if the tumefaction is large, it is impossible to distinguish it from a neoplasm, even on exploration.

Cancer of the right half of the colon usually exists about thirteen months before diagnosis is made and in forty-three per cent of the cases, metastasis to the lymph nodes has already taken place. Clinical manifestations vary widely depending upon the size, site, the form of the tumor, the presence or absence of ulceration, perforation, secondary infection, metastasis, etc. For instance, a growth of small size may cause obstruction very early if the ileocecal valve is involved, if not, obstruction occurs very late. The right half of the colon differs from the left half anatomically and physiologically. It is developed embryologically from the mid gut. It is supplied by the superior mesenteric artery and its function is absorptive, forty to fifty per cent of the water being absorbed in this part of the colon. The diameter is larger than that of the left side and the walls are thin, the contents are liquid and teeming with virulent organisms.

The growths are usually large and ulcerating affairs, often covered with nodules and are not inclined, as a rule, to encircle the bowel and cause obstruction. Because of the foregoing conditions the lesion is apt to go on to perforation or abscess formation. The symptoms are apt to be vague. Epigastric discomfort and local tenderness below the ribs or the right half of the abdomen that persist, are frequently early signs of carcinoma. They may complain of nausea, eructations, fullness after meals and lower abdominal distention. They are frequently diagnosed as either chronic appendicitis or cholecystitis.

The usual history of change in the bowel habit is apt to be absent. Intermittent attacks of diarrhea with periods of normal bowel movements with no tendency to constipation is often noted.

Secondary anemia without visible blood loss and loss of strength without explainable cause, may be the only two indications of malignancy of the right half of the colon. The hemoglobin may be as low as twenty-five to thirty per cent and one may suspect that a primary anemia exists, however, the anemia of malignancy is always of the secondary type, thus creating suspicion that either the stomach or right half of the colon is involved by a neoplasm and the patient should be x-rayed for differentiation. The degree of the anemia is greatest in cases of malignancy of low grade according to Clark. The cause of anemia is not very well understood but there is a definite iron deficiency. Anemia may be due to absorption of toxins because of the impaired selective absorption of the mucosa. The degree of anemia does not necessarily indicate whether or not the lesion is resectable, however, some feel that the size of the growth is in direct ratio to the degree of the anemia. Bright red blood is not apt to appear in the stools, but occult blood can nearly always be found. In approximately ten per cent of the lesions of the cecum, a tumor is found, either by the patient or on routine examination. Tumefaction is apt to occur early in this location. Loss of weight and cachexia are unimportant in early diagnosis.

X-ray findings in malignant lesions in the cecum and ascending colon may be confusing and early lesions are overlooked because the barium filled colon may over-lap and hide the lesion, particularly if the lesion is on the posterior wall.

If one is at all suspicious, repeated x-rays must be taken at intervals. In stout people it is difficult to palpate lesions in the cecum and ascending colon and difficult even in thin people to palpate lesions in the hepatic flexure. The size of the tumor is no index to its curability. Metastasis occurs late, so if diagnosed early the results of surgery are good. If no lymph nodes are involved, sixty-eight per cent

are apt to survive five years or more; if the lymph nodes and contiguous structures are involved this is reduced to thirty-seven and five-tenths per cent.

Segmental colitis may occur in the right half of the colon and is characterized by mild diarrhea, abdominal cramps, loss of weight, moderate anemia and frequently the presence of a mass in the right lower quadrant.

There may or may not be a rectal involvement. X-ray may simulate carcinoma but usually the filling defect is longer in extent, the mucosal pattern is lost and is smooth rather than irregular, the involved wall is thickened but relatively pliable and hyperirritability is usually present. Operation is indicated when the lesion shows a tendency to progress to a new territory or whenever the pelvic colon is threatened, because a healthy sigmoid must be present in order to do an ileosigmoidostomy with or without resection of the colon.

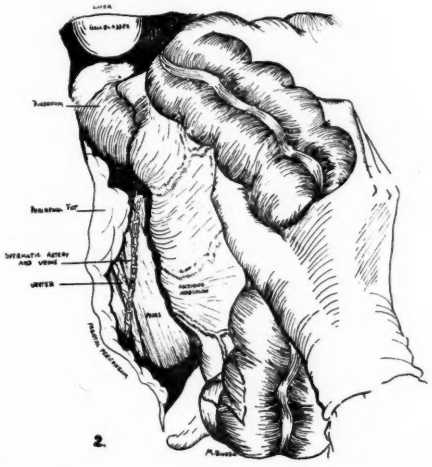


Fig. 2—Showing reflexion of colon medially.

Tuberculosis involving the right half of the colon is of two types: the hyperplastic type and the diffuse ulcerative type that follows advanced pulmonary tuberculosis.

The hyperplastic type which is the one which is apt to confuse us is rare, usually primary and no other tuberculous focus is evident. All the coats of the bowel are thickened by connective tissue proliferation and ulceration seldom takes place. It usually also involves the terminal ileum. Slight dyspepsia is apt to be present, with occasional attacks of pain and tenderness over McBurney's point. Irregularity of the bowels is not characteristic. A mass is present and often the appendix has been removed weeks or months before x-ray shows a filling defect of corrugated appearance with a palpable mass. The terminal ileum is involved and there is marked irritability of the intestine, the filling defect is longer, larger and less irregular than with carcinoma. Surgery is only indicated in tuberculosis to relieve acute obstruction or obstructed sequelae after the infection has been arrested. Operation may be a single or multiple stage resection. The results of resection of the hyperplastic type of tuberculosis are excellent. In all surgery of tuberculosis, streptomycin should be used preoperatively and postoperatively.

Actinomycosis of the cecum is not common although the ileocecal and sigmoid region are the portions of the gastrointestinal tract most likely to be affected. It is a chronic infectious disease caused by the fungus actinomyces and is characterized by the tendency to form abscesses, sinuses, granulation tissue and brawny leathery infiltration of the surrounding tissues. It may be acute or chronic. The acute type may be mistaken for appendicitis and in fact may be coincident with appendicitis. Clinically it may be impossible to distinguish it from appendicitis, except in those cases where abscess formation has taken place and the formation of a sinus which not only fails to heal, but the surrounding skin becomes indurated and has the characteristic bluish-red discoloration. Examination of the pus and finding of the ray fungus will confirm the diagnosis.

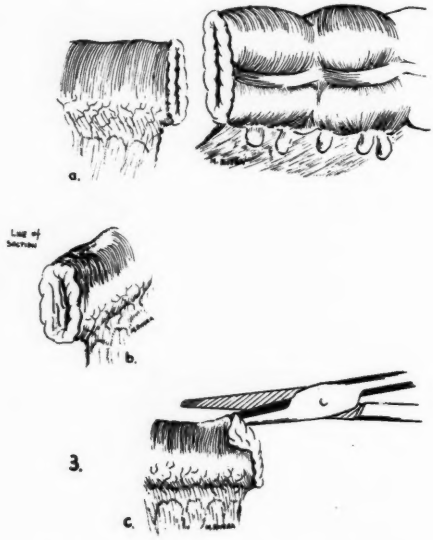


Fig. 3a—Disparity of lumen of terminal ileum and transverse colon. b. and c—Showing incision along antimesenteric border to increase the sizes of the lumen of the ileum.

The acute usually merges into the chronic variety before the diagnosis is made.

The chronic variety may present dull, vague pain occasionally followed by intestinal colic and irritability. Diarrhea may or may not be present. The first sign may be a slow-growing painless fixed tumor in the right side. These findings are not characteristic as the same things occur in hyperplastic tuberculosis and carcinoma of the cecum.

X-ray may give the clue to the diagnosis, however, in this region we have had one case recently that showed tumefaction which was fixed and hard in the right lower quadrant, with negative x-ray findings, which later broke down and formed an abscess. On exploration, the bowel was not involved and it was only by routine examination of the pus that the true cause, that is the ray fungus, was found. Usually it is not suspected until the formation of a sinus or sinuses that will not heal, and then the examination of the pus reveals the true condition.

In another case we thought we were dealing with an inoperable carcinoma of the cecum; the patient was closed after biopsy was done (the latter was negative) and it was only after discharging sinuses occurred that the condition was suspected and proven by microscopic examination of the pus.

The sulfa drugs and penicillin have proven very effective in actinomycosis but sometimes they only improve so far, then the fungus becomes resistant to the drug. Aureomycin apparently does much better and is the antibiotic of choice. Surgery is only indicated for the residual lesion after medical treatment.

Amebiasis occasionally causes a granuloma in the cecal, sigmoid or rectal region and particularly in the cecal region may form a palpable mass. These amebomas are due to invasion of the tissues by amebae with superimposed secondary infection. The characteristic x-ray picture, however, shows inflammatory induration with coning tumefactive defects, spasm and dilatation of the cecum and abnormal patency of the ileocecal valve. Of course the finding of the amebae in the stool clinches the diagnosis.

Nonspecific infective granuloma is a definite disease, the cause of which has not been ascertained and is always associated with inflammatory hyperplasia. It may be either the acute fulminating type or of the subacute variety. The stenotic type rarely, if ever, occurs in the right half of the colon. Symptoms of the first type resemble acute appendicitis for which it is usually mistaken and there is a rise in the white blood count and temperature. Tenderness in the right lower quadrant is present and possibly voluntary rigidity. If operation is done and this type of lesion is found and recognized, the abdomen is closed with or without drainage, depending on the experience of the operator.

The subacute type usually has a slow onset, varying from several days to several weeks, with remissions followed by exacerbations; they have local tenderness, rarely nausea and vomiting, with a slight rise in temperature. A mass develops and occasionally loss of weight and appetite with exhaustion may occur. As a rule no anemia is present except in cases that have a partial obstruction for some time. Usually there is a sense of well-being, which is absent in malignancy. The moon-shaped defect in the x-ray, plus the history, absence of anemia and feeling of well-being, suggest the diagnosis. Operation is not done as a rule if the diagnosis is made, unless obstruction is present, however, in some cases when the diagnosis is doubtful, even when the abdomen is opened, resection may be done either in a single or in multiple stages.

We have had one of these which followed three months after an appendectomy for a mild acute appendicitis, in a twenty-year old female, which may have been due as suggested by Mock, to ligatures and sutures that were not absorbed and acted as foreign bodies. This case was explored after a careful work-up, and, after satisfying ourselves at exploration that it was nonmalignant, she was closed and carefully watched. The mass slowly disappeared and at the end of three months she had completely recovered.

We also had two other cases which went on to abscess formation that were found to be due to foreign bodies. One due to perforation of the cecum by a tooth-pick on the medial side, and another by a chicken bone (fibula) which perforated the lateral side of the cecum. In these cases, removal of the foreign body and drainage caused a rapid subsidence of the tumor mass.

Appendicular abscess today is rarely confused with carcinoma of the colon. Benign tumors, particularly if they are single, may cause difficulty in diagnosis; they are—adenomas, fibromas, myomas, lipomas, angiomas, hemangiomas and cholesteatomas.

Appendicular abscess today is rarely confused with carcinoma of the colon. elevations to huge masses. Polyps are usually diagnosed by x-ray. Lipomas are rather rare, occurring about once in every 56,000 cases or an incidence of 0.002 per cent. The adenomas are usually classified as premalignant or early malignant tumors. Clinically most of the adenomas and about 72 per cent of the lipomas cause symptoms. These cases may present vague complaints and the tumor is found either by palpation or in a roentgenogram of the colon. Others may present bleeding and loss of weight, there is a tendency to intestinal obstruction particularly due to intussusception. Fifty per cent of the lipomas, according to Bacon, are in the right half of the colon. Diagnosis by x-ray usually indicates a polyp and translucency of the polyp after air injection suggests lipoma. The benign tumor can be excised, or enucleated after colotomy, if the diagnosis is certain. If the diagnosis is not definite, resection and anastomosis should be done.

Fig. 4—End-to-end anastomosis of ileum and colon.

Intussusception, particularly the chronic or the recurrent type, may be difficult to differentiate from carcinoma, actinomycosis or tuberculosis. X-ray with a barium enema usually makes the diagnosis. Very often it is associated with a tumor, either benign or malignant, which is the precipitating factor of the intussusception. Treatment consists in laparotomy, correction of the intussusception and resection of the lesion, if complicated by tumor.

Familial polyposis and chronic ulcerative colitis are mentioned because they involve the right half of the colon as part of the general process. The treatment in the former is total colectomy on account of the danger of malignancy. Ulcerative colitis may be treated medically or surgically, depending on the indications. In all operations on the right half of the colon, careful evaluation of the patient as a whole, must be done. Fluid and electrolyte imbalance must be corrected by adequate administration of water and minerals, either orally or parenterally. Anemia, which is so common in carcinoma in this region, must be corrected by multiple transfusions of whole blood. Blood volume estimates are very important because the ordinary red blood cell count often does not indicate the true reduction in total hemoglobin and total blood volume, consequently one may undertransfuse unless the blood volume estimates are done.

Hypoproteinemia is very apt to be present and should be corrected preferably by the oral administration of protein. Avitaminosis should be properly taken care

of by the administration of vitamins, particularly B and C, preoperatively. Sulfasuxadine, sulfathalidine or streptomycin should be given orally to lessen the bacterial content of the intestine, thereby decreasing the incidence of peritonitis. Vitamin K should be given in addition to these, because in decreasing the bacterial content of the bowel, one also decreases the formation of Vitamin K.

Where there is evidence of obstruction, either partial or complete, at the ileocecal valve or at any point beyond the ileocecal valve in the right half of the colon, *providing the valve is incompetent,* suction by means of a long tube will often decompress the bowel sufficiently to allow primary resection.

Resection of the colon may be done either in one stage or in multiple stages, although formerly it was the custom to do an ileocolostomy and resect the right half of the colon at a later date. Today, if the proper indications are present, we prefer to do a primary resection of the whole right half of the colon, the terminal ileum, with adequate removal of the mesentery, followed by immediate end-to-end anastomosis of the ileum to the colon.

Resection and immediate end-to-end anastomosis is the ideal operation, but should not be done if the bowel is not properly prepared. Primary resection and immediate anastomosis, either side-to-side or end-to-side, may also be done.

In multiple stage operations, the ileocolostomy, either lateral or end-to-side anastomosis is done first and the distal end of the ileum is closed and dropped back into the abdomen. Resection of the terminal ileum and right half of the colon is then done at a later date.

Again, if primary resection and immediate anastomosis is contraindicated, the Lahey-Mikulicz procedure is very valuable. This may be done very rapidly and is often indicated in the aged, those suffering from serious complicating disease, the very obese and where obstruction or pericolonic infection exists. The spur can be crushed in five to seven days.

With end-to-end anastomosis, the disproportion between the ileum and the colon is corrected by incising the antimesenteric border, thus increasing the size of the portion to be anastomosed. Obviously the anastomosis is of the open type, using fine chromic catgut for the inner layer and interrupted silk for the outer layer. The mesentery is carefully approximated, the raw surface left by the removal of the colon may or may not be peritonealized, depending upon the judgment of the operator and the amount of peritoneum available. It has been shown by Brunschwig that the peritoneum will usually regenerate to cover the raw surface. Drainage is not used as a rule.

If fixation of the colon, perforation, particularly with abscess formation which is so common in carcinoma in the right half of the colon, or obstruction exists, a two-stage operation should be done.

When obstruction of the right half of the colon exists, the procedure used will depend upon the site of the obstruction and the competency of the ileocecal valve. If the ileocecal valve is incompetent these cases can be decompressed by the use of the long suction tube and a single stage procedure can be done. If the ileocecal valve is competent and obstruction distal to it is present, then obstruction in a closed loop exists and prompt surgery is imperative. Primary resection and immediate anastomosis or a Lahey-Mikulicz procedure may be done if the patient's condition permits it. In those cases when the ileocecal valve is competent and the closed loop is sometimes tremendously distended, one may decompress the bowel by inserting a catheter into the ileum and through the ileocecal valve, with subsequent collapse of the distended loop. This makes it much easier and less dangerous to handle at operation. The competency of the ileocecal valve in case of

obstruction can be determined by the presence and location of gas and fluid levels in the colon and small intestine.

Postoperatively, fluid and electrolyte balance is maintained by intravenous administration of glucose and distilled water. Saline solutions are usually not given until 48 to 72 hours later. Transfusions are given if indicated. Decompression is maintained by the use of continuous intragastric suction or by the use of a long tube. In the latter case fluids can be given earlier because they are absorbed in the upper portion of the gastrointestinal tract. If the bowels do not move spontaneously by the third or fourth day, glycerin suppositories are given by rectum. The antibiotics—penicillin and streptomycin are used routinely after operation. As soon as fluids can be taken by mouth, sulfasuxidine is given orally. This serves, not only to decrease the danger of infection, but may also cause spontaneous movement of the bowels. Early ambulation is practiced.

## Summary

Lesions of the right half of the colon, both acute and chronic, have been discussed, the main problem is to differentiate these lesions from carcinoma, which should be constantly kept in mind.

Primary resection and end-to-end ileocolostomy is the operation of choice.

Routine use of chemotherapy and antibiotics, both preoperatively and postoperatively, have permitted more radical resection with greater safety and excellent end results.

Blood volume determinations have given us a means to estimate the amount of blood needed preoperatively. Adequate transfusion of whole blood before, during and after operation is absolutely essential.

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# PRURITUS ANI

## A Mechanistic Approach to Etiology and Treatment

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Pruritus ani, as every clinician knows, is a widely prevalent symptom complex. In its simplest terms, it is an inflammatory skin reaction, sometimes representing a somatic abreaction.

### Classifications

Various attempts at classifying pruritus ani have been made; for example, for cases in which no cause can be found: primary or idiopathic[1] or cryptogenic[2]; or again, chronic severe idiopathic intertrigo[3]. In cases in which one or more etiologic factors can be found, the condition is often referred to as secondary pruritus ani. Another investigator[4] distinguishes between true pruritus and false pruritus and believes that in false pruritus ani—a reflex nerve stimulus—the skin does not undergo the characteristic changes of true pruritus. I do not believe that clinical observations entirely support this view, for at least lichenification soon results in the course of the itch-scratch cycle regardless of the etiology[5].

Generally, pruritus ani is thought of quite simply as acute or chronic[6]. In the acute conditions there is redness, edema, excoriation, vesiculation, and sometimes weeping from the denuded areas. In chronic states there is lichenification, induration, pigmentations. In recalcitrant cases the skin may be whitish, smooth and glistening; or leathery and dotted with pigmented spots; or again, the skin may lie in deep folds radiating from the anal orifice[6].

### Etiology

It is not the purpose of this presentation to appraise the validity of each of the many etiologic theories that have been advanced as being the cause of pruritus ani. Any comprehensive outline of the causes and predisposing factors would still be incomplete from the point of view of some clinicians whose experiences have differed further. Indeed, one is reminded of the story of the three blind men and the elephant, as continuous reading of the literature and mounting personal observations reveal another and yet another suggested cause:

*Constitutional diseases* (nephritis, tuberculosis, and especially diabetes)

*Metabolic disorders* (thyroid dysfunction, anemia, and vitamin deficiencies)

*Dietary indiscretions* (excessive intake of carbohydrates, fruit juices, alcohol)

*Allergies* (to foods, to drugs, to soaps, fecal matter, discharges, dyes, or chemicals in toilet tissue, in underwear or soap; or, allergic id-reactions)

*Parasites* (monilia or, as it is now called, candida; trichomonas, threadworms, itch mites)

*Fungi* (miscellaneous types)

*Thermal causes* (warmth and associated perspiration)

* Read before the Third Annual Convention of the International Academy of Proctology, Atlantic City, N. J., June 7, 8, 1951.

*Mechanical irritations* (uncleanliness, improper anal hygiene; tight-fitting clothes)

*Psychogenic factors* (fatigue, worry, uncertainty, maladjustments, unresolved problems, masturbation—these, especially in the constitutionally predisposed person)

*Anal diseases* (hemorrhoids, fissures, crypts, ulcers, fistulae, papillitis, pectenosis, proctitis and colitis, constipation and diarrhea)

*Perianal skin diseases* (erythema, folliculitis, marginal eczema, herpes)

*Adjacent skin disorders* (cystitis, prostatitis, cervicitis, vaginitis, particularly an irritating vaginal discharge)

*Trigger Mechanisms:*—Some conditions cited as the etiologic factors often seem to serve rather as the trigger mechanisms. Among these may be listed the climacteric degenerative skin changes and mucous membrane dermo-hypersensitivity to mechanical and chemical irritation; mild pruritus associated with hemorrhoids and fissures; purulent discharge from trichomonas; irritating drugs used in the treatment of leukorrhea; sensitivity for any reason following hemorrhoidectomy or fistulectomy; secretion of mucus following the Whitehead operation for hemorrhoids; tight-fitting girdles or underclothes as primary irritation; simple allergies, such as to nail polish; constant humidity in the anal skin caused by an acid rectal mucus from prolapsing hemorrhoids, rectal prolapse, or atony of the external sphincter[3]; mucus from proctitis or colitis; tumors; parasites; anal and intergluteal intertrigo[3].

*Intensification of Pruritus:*—Van Studdiford[1] among others has made the point that there may, moreover, be produced an intensification of pruritus from a primary irritation or dermatitis venenata by self-treatment with local medication, suggested by advertisements or by friends, or actually prescribed by some physicians who forget that "one cannot burn the itch out because the [irritating] medication acts the same way that scratching does and the vicious cycle is simply perpetuated[1, 6]". Intensification likewise occurs through repetition of the itch-scratch cycle.

*Neurodermatitis as an Etiologic Factor:*—In my own most recent series of 41 cases, presently to be reported here, a neurodermatitis was the etiologic factor. There is a great deal of support to be found in the literature of psychiatry and of dermatology in considering neurodermatitis as causative. The pathogenesis of pruritus ani is in itself logically substantiating. It is recognized, however, that the desire to find an over-all treatment tempts each of us to proclaim the discovery of a single etiologic entity. There can be no such conclusion without dogmatism; thus, there can be no panacea but only pragmatic encouragement through the results obtained by a given rationale of diagnosis and a given regimen of treatment.

All the factors cited, and more, are doubtless sometimes causative; doubtless, too, those causes have been demonstrated with sufficient repetition to persuade a given investigator that he has found *the* specific cause; ergo, *the* specific treatment. There is no single cause. There is no single and absolute treatment. The causes are multiple; thus, each patient must be approached without preconception. Only when all factors have been taken into account can the best treatment for a given patient be determined[6].

## Fundamental Clinical Observations

Once these philosophical restraints on hobby-riding have been self-imposed, however, there remain, fortunately, a number of concrete observations by which

to narrow the etiologic concept and by which to simplify the approach to treatment. Certain empirical fundamentals emerge as common to the experiences of all objective clinicians:

1. Pruritus ani is exhibited either as a secondary manifestation of a local or distant pathologic process[4] or as a chronic idiopathic entity. As a secondary symptom complex, pruritus ani, if not arrested, may become a recalcitrant complication, more disturbing to the welfare of the patient than the original basic disorder.

2. As a chronic idiopathic disturbance, pruritus ani is of psychosomatic significance[6].

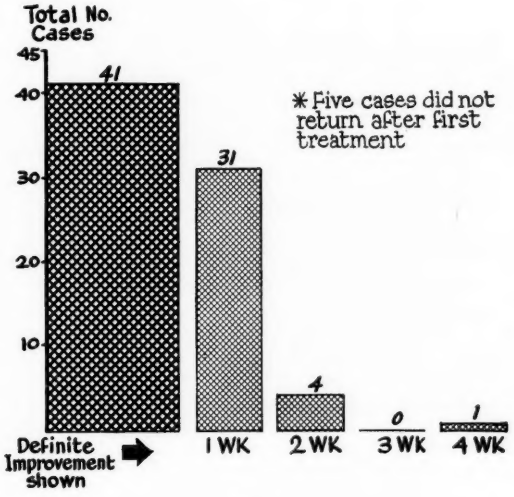


Fig. 1

3. Children and adults, men and women, any age group—all may be affected. The great bulk of patients, however, appear to lie between the two decades of 40 and 60.

4. Persons with thin skin are thought[7] to be more susceptible to pruritus ani; blondes more than brunets; men more than women, obese men more than slender; the comfortable and the rich more than the poor; the sedentary more than the wholesomely active[8]; the idle as well as the overly active more than the moderately busy.

5. In general, it may be said that most patients have been over-treated. Drastic procedures have too often been routinely attempted before simpler methods have been found wanting.

6. Itching cannot be controlled by counter-irritants.

7. It should be remembered that x-rays will not cure the patient. The "x-ray reputation" is not justified[9]. "The beneficial effect of x-rays is attained by anesthetizing the nerve endings in the [affected] area which relieves the pruritis long enough to interrupt the vicious cycle. . . ."[3].

8. Surgery "should be directed toward removal of sources of infection and the correction of obvious local disease. Surgery for any reason is contraindicated until the diseased and traumatized skin is healed and the host is symptom free" [1]. Routine removal of hemorrhoids relieves few patients; whereas hemorrhoid removal often acts as the trigger mechanism.

9. The patient must not be summarily dismissed as a neurotic. All patients with pruritus ani are highly nervous; but often the nervousness is the result of the pruritus ani and not the cause[5].

10. The condition may appear in a given case under a given set of stimuli but not in another case under the same trigger circumstances[4]. This is largely because there is a pretty well defined constitutional type, especially susceptible to pruritus

| DURATION | DEFINITE IMPROVEMENT SHOWN | | | |
|---|---|---|---|---|
| | 1 WK. | 2 WK. | 3 WK. | 4 WK. |
| 0-11 MOS. | 13 | 1 | | |
| 1-5 YRS. | 11 | 2 | | |
| 6-10 YRS. | 5 | 1 | | |
| 11-15 YRS. | 5 | | | |
| 16 YRS and OVER | 2 | | | |

Fig. 2

ani. Because of the importance of recognizing this type, the characteristics will be described separately at length.

11. Just as the etiologic factors are varied and manifold so are the suggested forms of treatment. Rosser[10] among others has stressed how illogical "are all attempts to use specific treatment on a mass basis for an undifferentiated group". As the etiology varies so must therapy be "individualized for each patient" [9]. No one form of therapy has been universally effective. "A unanimous rule for the efficacious treatment of pruritus ani does not exist"[3], and no therapeutic method is foolproof. The findings must indicate the therapy, whether it is to comprise the conservative measures which will suffice in the majority of instances or whether more heroic procedures are indicated in intractable cases—measures such as the Ball undercutting operation or its modifications; subcutaneous neurotomy; tattooing; injections of anesthetics in oil; or topical application of various drugs; or irradiation; or vaccine therapy; or histamine therapy, recently introduced in attempted treatment.

12. Fortunately, it is possible to establish the etiology in 90 per cent of the cases[3] and so to go on to treatment of the disclosed cause or causes.

13. Fortunately, too, most patients do recover completely following the institution of a conservative regimen, including, especially, proper hygienic measures.

14. The pathogenesis[5] of pruritus ani is best understood in terms of its vicious cycle which is one of itch→scratch→dermatitis→scratch. The skin of the anus and perianal region having a plentiful nerve supply is highly sensitive to stimuli of all kinds; hence, it is ready to respond to any irritation that will incite the itch-scratch cycle. This area is particularly accessible to every possible irritation, especially tactical, so that the vicious cycle may be set up quite by accident in anyone, and without fail in the constitutionally predisposed to whom the scratching may become an erotic equivalent, and the pruritus ani, becoming pleasurable however annoying, may become more difficult to eradicate as the hyperexcitability is further increased by repetition. Repetition, moreover, soon results in lichenification which in itself intensifies the itching[5]. In this process the threshold of susceptibility is steadily lowered[12] until the least and the lightest stimulus of any kind is enough to provoke the mechanism into motion. This is not to say that a single itch-scratch attack will inevitably incite a case of pruritus ani. But, if for any reason the scratch motion is employed often enough at short enough intervals in a futile attempt to relieve the repeated itch stimulus from any cause, then surely, attention will be fixed at the site of difficulty and the habit will become entrenched.

Tests demonstrating the physiology of itching[11] support the validity of this mechanistic explanation.

## Discussion of Constitutionally Predisposed Patient

As has been indicated, there is a preponderance of cases among patients having well defined and generally recognized[1, 6] constitutional characteristics. Such persons exhibit activity of a restless, exaggerated, driving nature coupled with concern over trivia[1]. They plunge into too many tasks during the day, usually accompanied by perfectionist attention to detail, but at night cannot relax and sleep. The pruritus, under the warmth of the covers and with the mind free to focus attention on the condition, becomes tormenting. Despite all the symptoms of nervous exhaustion and fatigue, such patients continue their overextended plans, and work with apparent energy, but are really presenting a classic picture of a type constitutionally predisposed to pruritus ani.

All psychogenic cases obviously fall into this group, as do many with an allergic cause, the mechanism of which must often be considered in terms of psychic conditioning.

The constitutional type usually has some unresolved problem to which he cannot adjust; therefore, he selects some organ or body system[1] as the "shock organ" of his personality. Such patients commonly develop any number of functional disorders but the anus and perianal region comprise a frequent and logical area of selection[6], because, like the vulva, it is essentially a zone of hyperesthesia and readily offers an erogenous substitute as well as an area of somatic abreaction[6]. That is why these patients present the most baffling cases and that is why I am dwelling on this type of patient at length. I am not presuming on the field of psychiatry, but as proctologists, we must not isolate ourselves in our specialty and forget the total personality whom we are attempting to treat or direct for further treatment.

It is these patients who are constitutionally predisposed to pruritus ani who present the most intractable and pronounced vicious circle, the pattern of which has been referred to and described. The cycle of itch→scratch→dermatitis→and scratch, with the parallel nervousness→irritability→sleeplessness→embarrassment often gives such marked pleasure from the scratching spell that the pruritic mechanism becomes suitable for the somatic abreaction[6] area and the scratching even becomes a masturbation equivalent.

TABLE I

| Name | Duration of Itching | Previous Prescription | Quantity X-ray Used | Definite Improvement | Cure | Follow-up | Neurodermatitis Elsewhere | Age of Patient |
|---|---|---|---|---|---|---|---|---|
| J. B. 7/27/48 | 6 mos. | Ointment | 103 r (in 2 doses) 7/27–8/3/48 | 1 wk. | | | 0 | 37 |
| A. B. 12/30/47 | 15 yrs. | Ointment | 132 r (in 3 doses) 12/30/47–1/13/48 | 1 wk. | 6 wks. | No itching after 1 yr. | 0 | 53 |
| J. B. 10/26/48 | 3 yrs. | Ointment and x-ray | 0 | No return | | | 0 | 42 |
| S. B. 7/13/48 | 2 yrs. | Ointment and x-ray | 57 r (in 3 doses) 9/10–9/27/48 | 1 wk. | 0 | Recurrent mild itching. Completely clear at times | Scrotum | 20 |
| S. B. 8/16/48 | 6 mos. | Ointment | 103 r (in 2 doses) 8/16–8/23/48 | 1 wk. | 2 wks. anal. 1 mo. vulvar | No itching 3 mos. later | Vulvar | 59 |
| N. C. 4/21/48 | 4 mos. | Ointment | 103 r (in 2 doses) 4/21–4/28/48 | 2 wks. | 3 wks. | | 0 | 53 |
| E. C. 1/27/48 | 12 yrs. | Ointment | 225 r (in 4 doses) 1/27–2/24/48 | 2 wks. | | | 0 | 43 |
| F. D. 7/16/47 | 2 mos. | Followed hemorrhoidectomy | 103 r (in 2 doses) 7/16–7/23/47 | 1 wk. | 2 wks. | | 0 | 47 |
| H. D. 7/27/48 | 15 mos. | Followed hemorrhoidectomy | 207 r (in 4 doses) 7/27–8/17/48 | 2 wks. | 1 mo. | | Scrotum | 69 |
| H. E. 6/17/48 | 1 yr. | | 75 r (in 1 dose) | 1 wk. | 2 wks. | | Scrotum | 46 |
| S. E. 11/5/48 | 25 yrs. | Followed op. for fistula-in-ano, had considerable x-ray | 0 | 1 wk. Marked in 3 wks. | | | Scrotal | 65 |

TABLE I—Continued

| Name | Duration of Itching | Previous Prescription | Quantity X-ray Used | Definite Improvement | Cure | Follow-up | Neurodermatitis Elsewhere | Age of Patient |
|---|---|---|---|---|---|---|---|---|
| W. E. 9/9/48 | 7 yrs. | Followed hemorrhoidectomy | 103 r (in 2 doses) 9/9–9/16/48 | 1 wk. | | Relapse (following thrombotic hem.) | 0 | 43 |
| | | | 141 r (in 4 doses) 10/9–11/11/48 | 2 wks. | | | | |
| C. G. 10/1/48 | 8 yrs. | Hemorrhoidectomy, considerable x-ray | 0 | 1 wk. | 1 mo. | | | 64 |
| P. G. 1/25/49 | 10 yrs. | Gyne. operation | 75 r | 1 wk. | 1 wk. | | Vulva | 56 |
| L. H. 10/30/48 | 8 mos. | Ointment | 113 r (in 2 doses) 10/30–11/6 | 1 wk. | 2 wks. | No itching 1 mo. after last visit | 0 | 33 |
| M. H. 5/18/48 | 3 yrs. | Ointment | 161 r (in 3 doses) (5/18–6/1/48 | 1 wk. | 3 wks. | | Scrotum | 52 |
| J. I. 3/4/47 | 2 yrs. | Due to fungus | 0 | 1 wk. | ? | Monilia infection? 3 yrs. Treatment—Desenex. | 0 | |
| A. J. 2/16/48 | 6 yrs. | Local injections, ointment, 75 r x-ray | 150 r (in 5 doses) 2/16–4/12/48 | 1 mo. | 2 mos. | No itching 3 mos. after last visit. Last oint. tar. | 0 | 28 |
| K. K. 7/6/48 | 1 mo. | Ointment | 150 r (in 4 doses) 7/6–7/27/48 | 1 wk. | | Relapse about 4 mos. later; returned 12/2/48 Itching minimal and only occ. | Vulvar-pubic area | 45 |
| | | After recurrence | 150 r (in 3 doses) 12/2/48–1/22/49 | 2 wks. | | | | |
| M. K. 12/23/47 | 7 mos. | Ointment | 113 r (in 2 doses) 12/23–12/30/47 | 1 wk. | 2 wks. | | 0 | 38 |

TABLE I—Continued

| Name | Duration of Itching | Previous Prescription | Quantity X-ray Used | Definite Improvement | Cure | Follow-up | Neurodermatitis Elsewhere | Age of Patient |
|---|---|---|---|---|---|---|---|---|
| E. K. 2/18/47 | 3 yrs. | 1 x-ray, ointment | 113 r (in 2 doses) 2/18–3/4/47 | ? | | Did not return after 2nd visit. (Has diabetes) | Vulva | 50 |
| C. L. 9/27/47 | 3½ yrs. | Ointment | 188 r (in 4 doses) 9/27–10/25/47 | 2 wks. | | Only occ. itch when last seen—11/8/47 | 0 | 25 |
| P. McC. 1/21/49 | 14 yrs. | Undercutting hemorrhoid | 150 r (in 3 doses) 1/21–2/3/49 | 1 wk. | 3 wks. | | 0 | 45 |
| H. M. 11/12/48 | 6 wks. | Ointment | 57 r (in 2 doses) 11/12–11/15/48 | 3 days | | | | 60 |
| F. O. 11/3/48 | 2 yrs. Severe 2 mos. | Ointment | 94 r (in 2 doses) 11/3–11/8/48 | 5 days | 2 wks. | | Scrotum (Recurrence of scrotalitis-treated and improved) | 39 |
| K. O. 12/14/48 | 9 mos. | Ointment | 75 r (in 1 dose) 12/14/48 | | | Girl friend reported 1 mo. later that there was no itching | 0 | 19 |
| J. P. 2/1/47 | 1 mo. | 0 | 150 r (in 3 doses) 2/1–2/15/47 | 1 wk. | 3 wks. | No itching after several years | 0 | 38 |
| L. P. 2/20/48 | Several years | Ointment | 94 r (in 2 doses) 2/21–2/28/48 | 1 wk. | 1 wk. | | 0 | 42 |
| J. R. 11/18/47 | Several weeks Due to | 0 fungus inf. Tinea C | 38 r (in 1 dose) 11/18/47 | 3 wks. | 1 wk. | No recurrence | 0 | 63 |
| D. R. 2/17/47 | 8 mos. | Injec. therapy Ointment | 113 r (in 2 doses) 2/17–2/24/47 | 1 wk. | 2 wks. | | 0 | 29 |
| R. S. 1/8/48 | 4 yrs. | Ointment | 132 r (in 3 doses) 1/8–1/22/48 | 1 wk. | | Itch minimal and only occ. to Jan. 1949, then recurred mildly | 0 | 31 |

TABLE I—Continued

| Name | Duration of Itching | Previous Prescription | Quantity X-ray Used | Definite Improvement | Cure | Follow-up | Neurodermatitis Elsewhere | Age of Patient |
|---|---|---|---|---|---|---|---|---|
| J. S. 4/24/47 | Many years | Ointment | 113 r (in 2 doses) 4/24–5/24/47 | 1 wk. | | | Scrotum | 61 |
| H. T. 12/28/48 | 3 yrs. | Ointment | 75 r x-ray (1 dose) | 1 wk. | | No itching for several months | 0 | 52 |
| R. T. 1/11/47 | 10 yrs. | Ointment | 75 r (in 1 dose) 1/11/47 | 1 wk. | | No itching until summer of 1948 when she contracted amebic dysentery | Vulva | 39 |
| | | | 150 r (in 3 doses) 11/27–12/11/48 | 2 wks. | | | | |
| N. T. 12/2/47 | 12 yrs. | X-ray, nerve cutting, hormones | None to anal area | 1 wk. | | | Vulva— did receive x-ray here | 44 |
| R. T. 7/6/48 | 5 yrs. | X-ray, hemorrhoidectomy | 57 r (in 2 doses) 7/6–7/13/48 | 1 wk. | 2 wks. | | 0 | 38 |
| L. W. 2/11/49 | 8 yrs. | Ointment | 150 r (in 3 doses) 2/11–3/7/49 | 1 wk. | | | 0 | 45 |
| H. Z. 7/22/48 | 2 wks. Due to | fungus inf. Tinea C | ruris 38 r (in 1 dose) | 2 days | 2 wks. | | 0 | |
| L. Z. 1/8/49 | 15 yrs. | Ointment | 75 r (in 1 dose) | 1 wk. | 2 wks. | | Scrotum, pubic area, right arm | 58 |
| M. P. 1/11/49 | 8 yrs. | Undercutting | | | | Pt. had psoriasis plus superimposed monilial infection | | |
| R. Z. 3/4/49 | 2 mos. | Ointment | 75 r (in 1 dose) | | | | 0 | 52 |

Psychoanalysts and psychiatrists have treated this aspect of pruritus ani extensively. Its intensive discussion is not within my province; but, I repeat, it is essential that proctologists be aware of the frequent psychogenic etiologic factor in order to obviate needless surgery and ineffectual treatment, and to give their patients imperative insight into this tormenting condition.

## Report of Cases

In cooperation with a member of the Department of Dermatology in the Clinic of the Mt. Sinai Hospital in Chicago, a series of 41 cases was treated in which the etiologic factor was a neurodermatitis.

The duration of itching varied from one month to 25 years. All but two of the patients had had previous treatment for the condition; or, pruritus had followed anal surgery. In 14 cases there was an associated neurodermatitis of the scrotum or vulvar-pubic area, and in one instance, of the right arm as well.

The ages of the patients ranged from 19 to 60, with ten in the decade from 40 to 50; nine between 50 and 60, and five, 60 or over.

A complete breakdown of details is given in the accompanying tables. The method of treatment is described herewith:

## Outline of Treatment in the Absence of Organic Disease[13]

*Principles of treatment:*—It is my present belief that the very mechanism of pruritus ani in its reaction to its many trigger stimuli logically determines the principles of treatment: (a) Internal pathologic processes which by their secretions incite itching must be corrected; (b) external stimuli must be prevented; (c) the condition of skin hypersensitivity must be decreased by all hygienic and sedative means; (d) the patient must be given insight into the causes and effects of the plaguing condition in order to secure the patient's cooperation in treatment.

*Procedure:*—1. There is to be no rectal manipulation, not even enemas, once treatment has been instituted. For that reason, all examinations to eliminate the possibility of organic disease must be completed before treatment is begun.

2. Recommend avoidance of all external stimuli which may start an itching spell. These include: (a) tight or roughly-woven clothing so as not to set up a mechanical irritation; (b) overheated bedrooms and bathrooms; (c) underwear containing cheap dyes; (d) cessation of all previously used self-medication.

3. Instruct for habit-training of only one soft bowel movement daily.

4. The use of toilet paper is contraindicated and is strictly forbidden. Cotton is to be used instead in a blotting rather than a rubbing motion.

5. There must be a complete revision of the cleansing procedure. Hygienic measures are detailed:

a) A large pan or basin of lukewarm soapy water is to be placed in readiness on the bathroom floor before bowel movement. The soap should be a mild one and only enough of it used to make the water milky.
b) After defecation the anus is *patted* lightly with a moistened pledget of cotton to remove gross dirt. This is done not more than once or twice.
c) The basin of soap solution is then used as a modified sitz bath (with legs sprawled), for 5 or 6 minutes.
d) The anus is then rinsed with clean warm tap water.
e) The anal cleft is dried by blotting with gentle pressure, either cotton pledgets or extra soft white cleansing tissues being used.

6. A mild soothing ointment is prescribed for routine use after cleansing and to be repeated at bedtime:

| | |
|---|---|
| Phenol | 0.3 gm. |
| Boric acid | 1.5 gm. |
| Zinc oxide | 15.0 gm. |
| Petrolatum | 15.0 gm. |
| Rose water ointment to make | 60.0 gm. |

If there is an exudative dermatitis, calamine liniment (N.F.) containing 0.25 per cent phenol is prescribed. If there is infiltration and lichenification of marked degree and the routine prescription does not give relief, then the following may be prescribed:

| | |
|---|---|
| Ichthyol | 0.9 gm. |
| Zinc oxide | 10.0 gm. |
| Petrolatum to make | 30.0 gm. |

*Only in exceptional cases,* the following may be tried:

| | |
|---|---|
| Crude coal tar | 1.5 gm. |
| Paste of zinc oxide (N.F.) to make | 30.0 gm. |

All antipruritics of higher concentration are to be avoided.

7. Mild skin dosages of x-rays are given with low voltage, ranging from 25 to 80 roentgens, not more than once weekly. These are not continued after several irradiations even if the pruritus persists.

8. A mild sedative, usually phenobarbital, is prescribed to be used three times daily: midmornings, midafternoon and at bedtime.

9. Because itching is always intensified in bed, the patient is urged to rise early and to do no daytime napping.

10. Finally and importantly, the patient must be made to understand that cure is impossible without his cooperation. Give a simple explanation of the mechanism of the vicious cycle so as to instill the importance of avoiding any rubbing or scratching or irritation. With insight the patient will cooperate and cures can usually be effected.

## Follow-up Results

The detailed results of treatment insofar as they are available are given in the accompanying tables. There was definite improvement in from two days to one month, with 27 showing definite improvement in one week. In nine cases cure was effected in two weeks; four in three weeks; the maximum period was two months.

Recurrences, to be sure, are always a possibility; but when the condition is demonstrated not to be irreversible and the vicious cycle is effectively broken, recurrence is less likely and less frightening, and both the patient and physician may be encouraged.

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13. The method of treatment outlined is essentially that advocated by the dermatologist Arthur L. Shapiro of the Chicago Medical School.

# ABSTRACTS FOR PROCTOLOGISTS

**STRICTURE OF THE RECTUM. A NEW TREATMENT. Robert Turell. Am. J. Surg. 81: 71-77, (Jan.), 1951.**

In this paper the technic of endothermic resection of diaphragmatic strictures of the rectum by means of the double loop, high frequency resector is described and illustrated. While this work is still in its beginnings, the early results have been encouraging enough to warrant the continuation of this method of therapy and its extension to the treatment of the tubular type of stricture. A final evaluation of the procedure must await a prolonged period of follow-up observation (at least two years). Although the feasibility and the technic of electrosurgical resection of rectal strictures by means of the author's instrument has been established, this procedure is still in the experimental stage. Six illustrative case reports are included in this presentation.

DONALD C. COLLINS

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**ROUTINE EXAMINATION OF THE LOWER BOWEL. RESULTS IN 500 ASYMPTOMATIC PATIENTS. Vincent T. Young. Am. J. Surg. 81:18-22, (Jan.), 1951.**

Sigmoidoscopic examination is performed on all new adult patients who undergo general examination at The Yater Clinic, Washington, D. C. Records of 500 patients who did not complain of symptoms pertaining to the lower bowel have been accumulated and the data regarding lesions of the lower bowel reviewed. One hundred thirty-eight patients (27.6 per cent) showed lower bowel lesions. Sixty of these were males. The distribution of these lesions is given.

DONALD C. COLLINS

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**ROLE OF CHLOROPHYLL IN PROCTOLOGY. Bernard Horwitz. Am. J. Surg. 81:81-84, (Jan.), 1951.**

Chlorophyll is a non-toxic, bland substance which should occupy a prominent place in the treatment of acute and chronic rectal infections and wound repair in general. The similarity of chlorophyll to hemoglobin suggests the reason for the effectiveness of the former as an aid to tissue healing. Chlorophyll does not possess the antibacterial potency of specific chemotherapeutic agents, but its value in healing cannot be overlooked.

Twenty per cent dissolved benzocaine in a bland water-soluble base provides a potent, safe, topical anesthetic. Combined with diffusible chlorophyll it becomes very valuable when the regeneration of tissue is indicated. Such a preparation has a definite place in preoperative and postoperative treatment of rectal conditions when relief of pain and pruritus and acceleration of healing are needed.

DONALD C. COLLINS

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**RELATION OF ASCORBIC ACID PLASMA LEVELS TO ANORECTAL CONDITIONS. A PRELIMINARY REPORT. William A. Scheffler. Am. J. Surg. 81:85-89, (Jan.), 1951.**

Low ascorbic acid plasma levels are encountered frequently in patients with some acute seasonally occurring anorectal conditions. This fact is most evident in early spring. Ascorbic acid content of the blood might be a significant factor in unexplained mucosal bleeding. Ascorbic acid medication appears to be a valuable adjunct in treating the conditions mentioned if the level is found low. The author believes that further investigation by other observers in different climatic sections of the country would be of value.

DONALD C. COLLINS

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**MULTIPLE POLYPOSIS IN AN INFANT OF FOUR MONTHS Harry W. LeFevre and Thomas F. Jacques. Am. J. Surg. 81:90-91, (Jan.), 1951.**

The authors state that this is the youngest case of multiple polyposis on record to their knowledge. With the evidence of the autopsy findings, together with the accompanying microphotographs, it is hard to conceive how these polyps could have attained such size in only four months. They believe, therefore, that these polyps must have been present prior to the birth of this child and that he was born with multiple polyposis. The child was six months of age when he expired.

DONALD C. COLLINS

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**RADICAL SURGICAL TREATMENT OF CHRONIC ULCERATIVE COLITIS. Wayne W. Flora. Am. J. Surg. 81:92-93, (Jan.), 1951.**

A case report is made concerning a twenty-eight year old, white, female patient. For more than nine years she had suffered from a progressive destructive infection of the large bowel causing loss of sphincter function, perineal body and part of the rectovaginal septum, necessitating the constant wearing of a perineal dressing. Contemplating marriage and being the mother of a two-year old daughter, her decision to submit to a three-stage operation of considerable magnitude involving a permanent ileostomy, is evidence of the unsatisfactory and burdensome existence

which she was no longer willing to endure. Such patients should be encouraged to accept the risks of necessary surgery in anticipation of cure from an incapacitating and life threatening disease before irreparable damage has been done.

DONALD C. COLLINS

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**MALIGNANT MELANOMA OF THE RECTUM. David N. Yaker. Am. J. Surg. 81:94-95. (Jan.), 1951**

A case report is made of a housewife, aged fifty-three years, who had a hemorrhoid and a rectal polyp removed surgically. Histologic study of the polyp revealed it to be a melanosarcroma. The patient died in slightly more than five months from a widespread metastatic melanosarcomatosis.

DONALD C. COLLINS

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**CARCINOMA OF THE ANUS, RECTUM AND SIGMOID. Frederick B. Campbell and William C. Schaerrer. Am. J. Surg. 81:121-126, (Jan.), 1951.**

Three hundred consecutive private cases of anorectosigmoid carcinoma are reviewed for the period May, 1942 to March, 1950. Recent trends in the choice of operative procedures are shown by a comparison of three series of 100 cases each. Mortality and complications are reviewed. Finding and removing polypoid lesions of the colon is emphasized. An improved colostomy stoma by secondary reconstruction is described.

DONALD C. COLLINS

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**CARCINOMA OF THE COLON. Alvin Baldwin, Jr., Am. J. Surg. 81:135-138, (Jan.), 1951.**

The morphology of the right and left colon has much to do with the difference in the type of carcinoma encountered in the two sides. Early diagnosis is essential to increase the five-year survival rate. Management of carcinoma of the colon is discussed. The widest possible resection in carcinoma of the colon and lesions which are partly or completely below the peritoneal reflection should be performed. Management of carcinoma of the colon and rectum will depend on the presence or absence of obstruction, fixation and infection. Each must be dealt with separately. Lastly, the location of the tumor will likewise determine the procedure to be followed.

DONALD C. COLLINS

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**CONTRACTED ANAL CANAL. David C. Ditmore. Am. J. Surg. 81:67-70, (Jan.), 1951.**

Contraction of the anal canal may be classified according to pathologic conditions as of three general types. Types 1 and 2, which usually follow some operative procedure, are the subject of this paper. Diagnosis depends upon the history of previous operation, continued pain, increasing constipation and a sensation that the anal ring is too small. Complete relaxation under adequate anesthesia may be necessary before the exact type of contracture is ascertained. Pectenosis and coccygodynia which are not so well-known, are discussed in somewhat greater detail than other conditions to be differentiated from postoperative anal contraction. Postoperative anal contraction is a pathologic condition from which the patient suffers almost pathognominic distress. Cure may be expected following competent surgery and adequate postoperative care.

DONALD C. COLLINS

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**HYDROGEN PEROXIDE PROCTITIS. R. E. Pumphrey. Am. J. Surg. 81:60-62, (Jan.), 1951.**

There is no doubt that enemas using hydrogen peroxide solutions are effective in relieving fecal impactions. It is apparent, however, that the strength of the solutions used must be closely supervised in order to avoid complications.

The author states: "In my experience no more than 30 c.c. (1 ounce) or at the most 60 c.c. (2 ounces) of a U. S. P. solution of hydrogen peroxide (3 per cent) to 500 c.c. (1 pint) of water should be used." It should also be taken into consideraiton that varying strengths of hydrogen peroxide are available on the shelves of druggists. Two case reports demonstrate the harmful results of using strong solutions or even U. S. P. full strength of hydrogen peroxide for relieving fecal impactions. Both patients had severe ulcerative proctosigmoiditis following the administration of such enemas. Healing varied from three weeks to eight months after such vigorous enemas.

DONALD C. COLLINS

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**EVALUATION OF CHLOROMYCETIN AS AN ADJUNCT TO THE SURGICAL MANAGEMENT OF LYMPHOGRANULOMA VENEREUM AND SEGMENTAL ULCERATIVE COLITIS. Robert J. Rowe. Am. J. Surg. 81:42-54, (Jan.), 1951.**

The need for a specific therapeutic agent for lymphogranuloma venereum and nonspecific ulcerative intestinal disease has been evident. Certain virucidal properties of chloromycetin suggested that it would be of value in this capacity. Thirty-two patients with lymphogranuloma venereum and four patients with nonspecific ulcerative intestinal disease have been treated with

chloromycetin. Patients have been classified in four categories: nodular ulcerative proctitis (three patients), ulcerative proctitis with stricture (eleven patients), acute genital and inguinal lesions (eighteen patients) and nonspecific ulcerative intestinal disease (four patients).

Although inconclusive at this time, results indicate that chloromycetin has a specific activity against the virus-like agent of lymphogranuloma venereum. It shows promise of effecting a cure in the acute cases and palliation in the moderately advanced rectal lesions. Its use may preclude extensive surgical procedures such as abdominoperineal resection except in the most advanced rectal strictures. In the latter group it should be of value as an adjunct in preparation for resection. Results of treatment in the four patients with ulcerative intestinal disease indicate that chloromycetin may be of therapeutic value, but it probably has no effect on the causative agent of the diseases.

DONALD C. COLLINS

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**CONTROL OF HEMORRHAGE FOLLOWING ANORECTAL SURGERY. M. E. Rolens. Am. J. Surg. 81:28-30, (Jan.), 1951.**

Attention is called to the potential severity of postoperative anorectal hemorrhages. The diagnosis and causes are briefly noted. A simple and effective method of control is described using a three foot strip of once-folded 4½ inch wide absorbent sterilized gauze. A heavy linen mattress suture is passed through this packing to fold the gauze back and forth in an accordian-like fashion. After the rectum has been cleansed, this pack is threaded through the speculum and the rectum is snugly filled with this gauze. The speculum is then removed, while the pack is held in place with the forceps. The external part of the pack is then pushed against the anal margin with one hand while the other pulls on the free ends of the linen suture. When the tension is snug, the suture is tied. This creates a dumbbell-shaped pack. The inner sphere of the dumbbell exerts a firm pressure on the lower part of the rectum and the upper portion of the anal canal. The outer sphere exerts firm pressure on the external margin. Since there is only one strip of gauze in the anal canal, the pack causes very little discomfort. After the pack is in place a blood transfusion is started as soon as possible. This method is applicable only to those hemorrhages having their origin in the lower portion of the rectum, below the lower rectal valve.

DONALD C. COLLINS

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**MUCOID CARCINOMA OF THE RECTUM. H. D. Trimpi and H. E. Bacon. Cancer 5:597-609, (May), 1951.**

Mucoid carcinomas are adenocarcinomas that secrete mucus. In Grades I and II extracellular mucus is prominent, while signet-rings make up Grades III and IV. As symptoms are seldom present until widespread metastases occur, prognosis for cure is not favorable. Palliative surgery usually gives many months of ease and freedom from symptoms.

HAROLD NEIFELD

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**ADENOMATOUS POLYP OF THE RECTUM WITH SURFACE CARCINOMA. B. W. Warner. N. Y. State J. Med. 51:1640-1641, (July 1), 1951.**

The author reports the removal of a rectal polyp with carcinoma of its surface but no invasion of its base by an electric cutting current.

HAROLD NEIFELD

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**FACTORS RELATED TO AVOIDING COLOSTOMY FOR LESIONS OF THE RECTOSIGMOID. R. R. Best. J. Internat. Coll. Surgeons 15:352-356, March), 1951.**

Because the downward spread (more than 2 cm. below the palpable margin of the tumor the so-called margin of safety) in malignant lesions at the rectosigmoid levels is exceedingly infrequent, Best does not believe that 5 inches (13 cm.) of rectum below this margin of safety and the sphincter mechanism should be sacrificed.

HAROLD NEIFELD

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**USE AND ABUSE OF ANTIBIOTICS IN TREATMENT OF LOCALIZED PILONIDAL AND PERIRECTAL SUPPURATIONS. R. Turrell. N. Y. State J. Med. 51:619-620, (March), 1951.**

While antibiotic therapy against susceptible organisms is useful in the treatment of some cases of pilonidal or perirectal cellulitis, Turrell advocates early, adequate surgery in established localized suppurations in the sacrococcygeal, perianal or perirectal regions.

HAROLD NEIFELD

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**INCOMPLETE ROTATION OF THE CECUM WITH VOLVULUS OF THE MIDGUT. L. Salvati and C. G. Hanson. J. Internat. Coll. Surgeons 15:743-747, (June), 1951.**

The authors report a case that was operatively treated with a successful result.

HAROLD NEIFELD

# BOOK REVIEWS FOR PROCTOLOGISTS

**SURGERY OF THE STOMACH AND DUODENUM, Claude E. Welch, Associate Visiting Surgeon, Massachusetts General Hospital; Clinical Associate in Surgery, Harvard Medical School, 349 pages, 79 illustrations, The Year Book Publishers, Inc., Chicago, 1951. Price $8.50.**

This handbook of operative surgery is the most useful handbook of its type known to this reviewer. It is practical for the younger surgeon particularly, but should not be ignored by the established surgeon.

The technics illustrated are those employed at the Massachusetts General Hospital on the ward service.

It is interesting to see the use of transthoracic approaches. The various steps in surgical technics are accurately and fairly developed by excellent black and white line drawings.

The anatomy of the stomach and duodenum is first illustrated, followed by a discussion of pre- and postoperative treatment and anesthesia, as well as the special instruments required.

This text is recommended without reservation for all surgeons in training.

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**THE PHARMACOLOGIC PRINCIPLES OF MEDICAL PRACTICE, John C. Krantz, Jr., Professor of Pharmacology, School of Medicine, University of Maryland; Secretary of the General Committee of Revision of the United States Pharmacopoeia 1940-50, and C. Jelleff Carr, Associate Professor of Pharmacology, School of Medicine, University of Maryland; Auxiliary Member of the Revision Committee of the United States Pharmacopoeia 1940-50. 1116 pages, 94 figures. The Williams & Wilkins Co., Baltimore, 1951, Price $10.00.**

This very excellent second edition of the Krants and Carr text discusses the pharmacotherapeutic action of drugs as they are used in the treatment of disease. The chapter on the antibiotics is up-to-date, including aureomycin, chloraphenicol and terramycin.

The second edition contains two new chapters, one on Rickettsial Diseases and one on Chemotherapy of Tuberculosis. There is also a new chapter on Antimotion Sickness Drugs, the treatment of various forms of arthritis, and on ACTH and Cortisone.

Inasmuch as the text is intended chiefly for the medical student and the clinician, its major emphasis upon the action of drugs is commendable.

This text is well written, highly authoritative, and up-to-date.

The proctologist will find much of value in the general sections as well as in the specific drug therapies. The Chemotherapy of Amebiasis and Helminthiasis is particularly well discussed. With the present stress upon psychosomatic medicine the proctologist will need to know more about the response of the central nervous system and the autonomic nervous system to drugs. Both these subjects are well covered. There is also a separate chapter on the effect of drugs upon the gastrointestinal tract.

This book may be recommended without reservation.

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**CLINICAL PARASITOLOGY, Charles Franklin Craig, M.D., M.A. (Hon.), Sc.D. (Hon.). F.A.C.S., F.A.C.P., D.S.M. Late Colonel United States Army, Formerly Director, Army Medical School, and Assistant Commandant, Army Medical Center, Washington, D. C.; Emeritus Professor of Tropical Medicine in the Tulane University of Louisiana, New Orleans, Louisiana, and Ernest Carroll Faust, M.A., Ph.D., The William Vincent Professor of Tropical Diseases and Hygiene and Head, Division of Parasitology, Department of Tropical Medicine and Public Health, The Tulane University of Louisiana, New Orleans, Louisiana; Consultant to the Surgeon General, U. S. Army; Consultant, U. S. Public Health Service. 1032 pages, 326 figures. Lea & Febiger, Philadelphia, 1951, Price $12.00.**

The fifth edition of the classical text brings the work of Craig and Faust up-to-date. The book is completely revised.

The clinician will find a complete discussion of the newer drugs for the treatment of malaria, and filariasis. New chapters on Insecticides will be found, and a new section on Epidemiology.

With the increasing movement of civilian and troop personnel from one part of the world to another there is great need that the physician be oriented in the field of parasitology. This is particularly true for the proctologist.

It is this reviewer's opinion that every proctologist should be a competent parasitologist.

This text will provide the answer to the proctologist's need for further information in the field. Etiology, pathology, symptomatology, diagnosis, treatment and control of every type of parasitic infestation is fully and authoritatively discussed.

The technical appendix is particularly valuable in that technics and diagnostic aids are outlined.

The medical student, the general clinician, and the proctologist will find this text of great value.

**AN ATLAS OF ANATOMY, J. C. Boileau Grant, M.C., M.B., Ch.B., F.R.C.S. (Edin.), Professor of Anatomy in the University of Toronto. Third Edition, 637 illustrations. The Williams & Wilkins Co., Baltimore, 1950, Price $12.00.**

The third edition of this very fine Atlas of Anatomy is even better than the first two. There are over 70 new illustrations. These include serial dissections of the pelvis and perineum, the palate and back of the neck, and the middle ear, as well as the arterial supply to the head of the femur, the knee, the walls of the nose, the veins of the axilla and the front of the thigh, the bronchial tree, the bronchopulmonary regions, and variations in the pancreatic ducts.

All illustrations are very skillfully done, and of considerable technical value.

All structures of the human body are illustrated by region, just as they would be found during dissection. The anatomy is shown with considerable accuracy, having been drawn both from the original specimen and photographs. Thus, the student will find this text of greater value than the greatly exaggerated type of illustrations hitherto available. The proctologist will be particularly pleased with the dissections of both male and female pelvis and the rectum. Illustrations to show the newer concepts of the anatomy of the sphincter musculature will be found, and are excellently prepared.

This book can be recommended for the student and for the surgeon who wishes to keep his basic anatomy before him at all times.

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**THE PHYSIOLOGY AND PATHOLOGY OF HEMOSTASIS, Armand J. Quick, Ph.D., M.D., Professor of Biochemistry, Marquette University School of Medicine. 188 pages, 18 illustrations. Lea & Febiger, Philadelphia, 1951, Price $4.00.**

The many recent advances in the study of blood coagulation and hemostasis are incorporated in this splendid little volume. The coagulation reaction is well described in the first clinical section of the book. Dr. Quick's theory of coagulation provides an interesting explanation for the extension of a thrombus in phlebothrombosis. Thus, a technic of prophylaxis is developed.

The author states that "the fibrin clot does not function primarily as a static mechanical plug, but rather as a resrvoir of entrapped platelets and an adsorbent of thrombin whereby it localizes thrombosis and maintains sustained hemostasis by continuous liberation of the vasoconstrictor principle."

Hemostasis is described as both a coagulation reaction of the blood and a vascular response, mediated through the platelets.

The first 100 pages combines theoretical and clinical material, resulting in a useful diagnostic and therapeutic classification for hemorrhagic diseases. The second half of the book presents 21 laboratory tests that have proven useful to the author and others in the clinical study of hemorrhagic diseases.

Every surgeon is concerned with clotting and hemostasis. It is important to the specialty surgeon, the proctologist, and the general surgeon, to have this material at his fingertips. The clinical application of this type of writing will be very valuable to the surgeon.

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**SURGICAL PRACTICE OF THE LAHEY CLINIC, Members of the Staff of Lahey Clinic, Boston. 784 illustrations on 509 figures. 1,014 pages. W. B. Saunders Co., Philadelphia, 1951, Price $15.00.**

This excellent volume presents surgical practices which have become standardized in the Lahey Clinic. As such, we must realize that the procedures described represent the practices considered best for the members of this Clinic. This does not necessarily mean that they are superior to others.

Dr. Lahey stresses this in his preface, and it is important to recognize this "limitation".

The general surgeon will find much of interest throughout the volume. The section on the Thyroid Gland and Neck is particularly well written and valuable. All sections are well illustrated and all technics are described in sufficient detail for the practicing surgeon.

The proctologist will find the section on the small intestine, colon, sigmoid and rectum, of particular value. The indications for surgical intervention in ulcerative colitis are well presented and worthy of careful study.

This section includes a discussion of resection of the small intestine, the management of chronic regional ileitis, technics of ileostomy, polyps of the colon and rectum, surgical treatment of polyps of the large intestine, primary resection of the colon, prognosis of carcinoma of the colon and rectum, the surgical therapy of diverticulitis and its complications, abdominoperineal resection for carcinoma of the rectum, and pruritus ani. All subjects are well discussed and illustrated.

It is good to see emphasis on psychogenic factors in pruritus ani. The authors have had no experience with the Cantor tattoo-neurotomy technic, and little experience with local anesthetic in oil preparations. This is unfortunate, and weakens the evaluation of therapy.

The text concludes with an excellent section on anesthesia, and a particularly valuable description of experiences with cardiac arrest. Again it is disappointing to find no discussion of caudal analgesia.

This reviewer would consider this text a classic in the presentation of American surgical practice.

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**DISEASES OF THE ENDOCRINE GLANDS, Louis J. Soffer, M.D., F.A.C.P. Associate Attending Physician and Head of the Endocrine Research Laboratory and Clinic, The Mount Sinai Hospital, New York City; Assistant Clinical Professor of Medicine, Columbia University. 1142 pages, 88 illustrations and 3 colored plates. Lea & Febiger, Philadelphia, 1951, Price $15.00.**

The role of the endocrines in body physiology, and deviations from normal physiology, has become increasingly clear during the past 10 years. The work of Selye and others, particularly on adrenal and pituitary hormones, has assumed a major significance in clinical medicine.

Thus, a modern text on endocrinology becomes essential in the library of every clinician, regardless of his specialty. Even the proctologist will find it necessary to study endocrinology in order to evaluate his patient's colon physiology and pathology, especially in ulcerative colitis.

This very excellent text is thus a welcome addition to any library. It is, perhaps, one of the most complete texts on the subject now available. The contents are divided generally into a section on The Hypophysis, The Adrenals, The Gonads, The Thyroid, The Thymus, and Parathyroids, and a final section on Hypoglycemia and other pancreatic dysfunctions. This division makes for ready reference.

In each section there is adequate consideration of embryology, anatomy and histology, as well as physiology and pathology. Clinical syndromes are carefully described.

The section included as an appendix, describing laboratory tests of endocrine function, deserves special mention.

Generally speaking this is a very well written, carefully detailed and authoritative text. The clinician will find it especially valuable from his own view-point of diagnosis and therapy.

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**CLINICAL HEMATOLOGY, Maxwell M. Wintrobe, M.D., Ph.D., Professor of Medicine and Director, Laboratory for the Study of Hereditary and Metabolic Disorders, University of Utah, College of Medicine, Salt Lake City, Utah; Formerly Associate in Medicine, Johns Hopkins University, Associate Physician, Johns Hopkins Hospital, and Physician-in-charge, Clinic for Nutritional, Gastrointestinal and Hemopoietic Disorders, Baltimore, Maryland. Third Edition, 1048 pages, 220 illustrations and 17 plates, 13 in color. Lea & Febiger, Philadelphia, 1951, Price $12.50.**

The third edition of the classical text book shows evidence of careful revision. New sections cover Cytochemistry, Heredity in Blood Disorders, and Splenic Puncture. The volume, generally, has been brought up-to-date, and the relationship of the hemopoietic disorders to endocrine physiology and pathology is described.

This book is authoritative and complete. Emphasis throughout is on the necessity for accurate diagnosis and specific therapy. Detailed consideration is given to laboratory procedures as they apply to office practice of the average general physician.

The illustrations are excellent and highly diagnostic. However, this reviewer would like to have seen more colored plates.

The proctologist will want at least one good hematology text book in his library so that he might evaluate the different types of anemia, to differentiate them from the secondary anemia of intestinal bleeding. There is consideration given to sprue, idiopathic steatorrhea and celiac disease. The macrocytic anemia of intestinal strictures and anastomosis, as well as that associated with chronic dysentery and regional ileitis is described.

The emphasis on diagnosis and therapy will be welcomed by every practitioner. This text is highly recommended.

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**CLINICAL TROPICAL MEDICINE, R. B. H. Gradwohl, M.D., Editor-in-Chief, Luis Benitex Soto, M.D., and Oscar Felsenfeld, M.D., editors, 1,647 pages, 473 illustrations and 6 color plates. The C. V. Mosby Company, St. Louis, 1951, Price $22.50.**

This is one of the best texts on clinical tropical medicine to reach this reviewer. It is well edited, and authoritatively written by 57 authors. The book is thus a well written record of the clinical experience of the writers, rather than a mere collection of literature.

Stress is placed upon nutritional, transmissable and climatic diseases.

The text is well illustrated throughout, and many of the illustrations are original.

The proctologist should have this volume in his library. Indeed, it should be in the library of every internist. However, the proctologist will be called upon more and more, with current international conditions, to make a diagnosis and supply the proper therapy for tropical diseases. The major manifestations of these diseases is often in the intestinal tract. The intestinal diseases caused by the protozoa, especially amebic dysentry, should be well understood by the proctologist.

The text includes an excellent chapter on amebic dysentery, useful clinical material on lymphogranuloma venerum and an excellent chapter on granuloma inguinale. The proctologist should also be acquainted with salmonellosis. He will find this well described in this excellent text.

Diseases caused by the various types of worm infestation are covered in a very thorough fashion. There is a special chapter on laboratory methods in the diagnosis of helminthiases.

From every point of view this text can be unhesitatingly recommended.

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**THE SPECIALTIES IN GENERAL PRACTICE, Edited by Russell L. Cecil, M.D., Professor of Clinical Medicine, Emeritus, Cornell University Medical College, New York City. 818 pages, 470 illustrations. W. B. Saunders Co., Philadelphia, 1951, Price $14.50.**

This book will probably become a classic reference book for the general practitioner. The volume covers the common specialties, from proctology to psychiatry. It is well written, well illustrated, and covers the major conditions safely treated by the general practitioner who does not have available to him, for immediate reference, the specialist in various fields.

Indeed, with the present trend being somewhat away from specialization and toward the re-establishment of the family physician, this book fills a need.

It is one of the few books written by specialists for general practitioners.

The first chapter deals with minor surgery, and covers the common conditions well.

Succeeding chapters describe orthopedic surgery, fractures and dislocations, urology, diseases of the anus, rectum and colon, gynecology, obstetrics, pediatrics, opthalmology, diseases of the nose and throat, diseases of the larynx, bronchi and esophagus, otology, dermatology and syphilology, and finally psychiatry.

The proctologist, of course, will be particularly interested in the section on the anus, rectum and colon. In this section the general practitioner is brought to the realization that he should at least examine the lower bowel. The average general practitioner who has a proctologist available for consultation will not wish to go beyond this stage. However, for those who desire to do their own surgery, the chapter indicates technics and some of the pitfalls. The general practitioner who wishes to do his own surgery, however, should also consult a more extensive reference work, such as *Ambulatory Proctology*, etc.

From every point of view this book may be considered a valuable addition for the general practitioner's library.

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**PLASTIC AND RECONSTRUCTIVE SURGERY, A Manual of Management, Ferris Smith, M.D., F.A.C.S., Consultant in Plastic Surgery Blodgett Memorial Hospital, Grand Rapids, Michigan. 895 pages with 592 figures. W. B. Saunders Company, Philadelphia, 1950, Price $15.00.**

This is one of the finest texts in the field. The illustrations throughout are very fine, complete, and well selected. The text is well written, authoritative, and covers the matter in a general and as well as a specific fashion.

The procedures selected indicate sound judgment and mature consideration. As a guide for the general surgeon who wishes to attempt a plastic reconstruction, for the experienced plastic surgeon, or for the specialty surgeon who wishes to operate in conjunction with a plastic surgeon, the text is very valuable.

However, there is nothing offered for the proctologist. This later statement must be modified in terms of Chapter 4 on Operative Procedure and Treatment. The very general material of this chapter can be applied in any field, including proctology. Indeed, it is valuable for the proctologist, and for any other specialty surgeon, to know the general principles of plastic surgery.

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**A TEXTBOOK OF MEDICINE, Edited by: Russell L. Cecil, M.D., Sc.D., Professor of Clinical Medicine, Emeritus, Cornell University, New York. Robert F. Loeb, M.D., Bard Professor of Medicine, Columbia University, New York. Associate Editors: Alexander B. Gutman, M.D., Professor of Medicine Columbia University, New York; Walsh McDermott, M.D., Associate Professor of Medicine, Cornell University, New York; Harold G. Wolff, M.D., Associate Professor of Medicine (Neur.), Cornell University, N. Y. 8th Edition, 1627 pages, 204 figures, 40 tables. W. B. Saunders Co., Philadelphia, 1951, Price $12.00.**

The 8th edition of this classical text book continues to offer authoritative presentations of the diseases of internal medicine. The coverage is clinically adequate.

In keeping with the present trend, there is particular emphasis upon the biochemical and psychologic aspects of disease.

The proctologist will be especially interested in the sections on lymphogranuloma venereum, tuberculosis of the alimentary tract, amebiasis, schistosomisasis and other metazoan infections, the sections on diarrhea; constipation, irritable colon, diverticula, ulcerative colitis, regional ileitis and intestinal neoplasms.

All sections are well written, clinically adequate, and sufficiently valuable for immediate application.

A brief section on psychosomatic medicine and another on the psychoneuroses may be read with profit by all specialists as well as by the general practitioner.

In general, this text may be recommended to all clinicians.

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**APPLIED DIETETICS, by Stern, Revised by Helen Rosenthal, B.S., Chief of Frances Stern Food Clinic, The Boston Dispensary, Assistant in Medicine, Tufts College Medical School; Pearl C. Baker, B.S., Former Associate, Frances Stern Food Clinic, The Boston Dispensary, and Wilma A. McVey, M.D., Assistant in Medicine, Tufts College Medical School. 293 pages, The Williams & Wilkins Company, Baltimore, 1949, Price $5.00.**

The public is becoming more and more aware of the role played by food in their general health. Thus, they are asking for diets and dieteric instructions in the treatment of their illnesses.

This very excellent text provides the answers for the busy clinician.

The text is divided into a section covering the daily food requirement of the body, followed by an excellent chapter on the construction of the normal diet. The therapeutic diet is then discussed. The remainder of the book is devoted to environmental factors that influence the effectiveness of the diet, education of the patient on the normal diet and on the therapeutic diet. This is followed by tables to simplify computation of the diet, dietary outlines and typical diets and menus.

The proctologist will be particularly satisfied with the sections relating to diets in colon and intestinal disease. There are excellent diets for spastic constipation and ulcerative colitis.

The book is well written and complete. Although published in 1949 there is very little to be added at this date.

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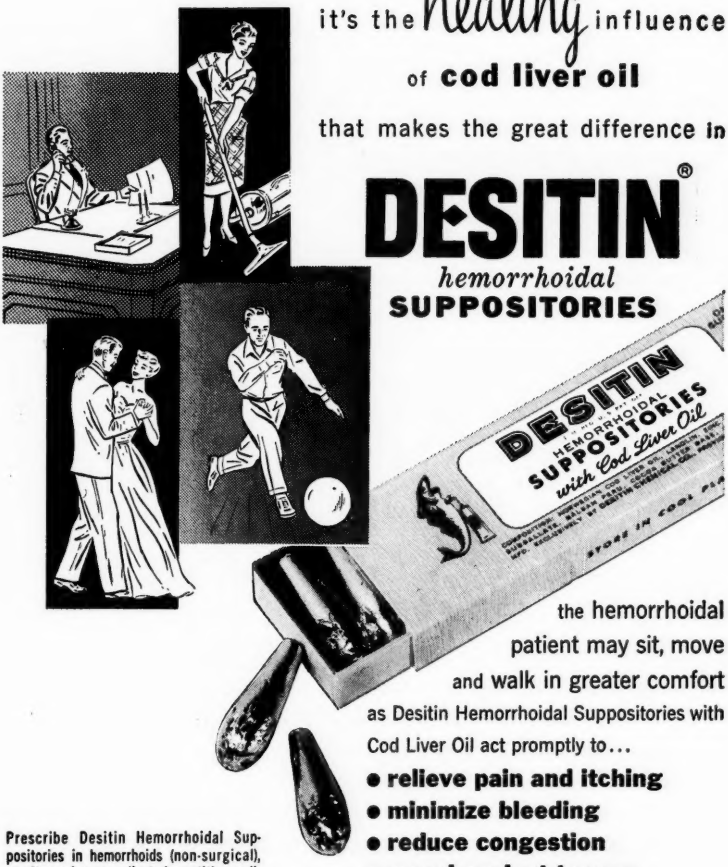


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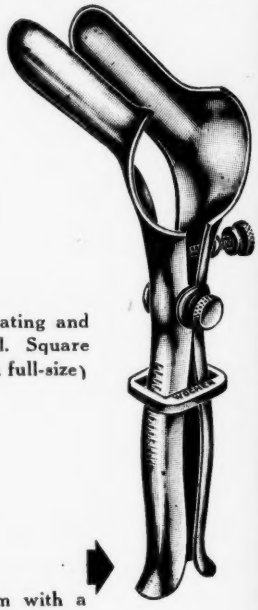

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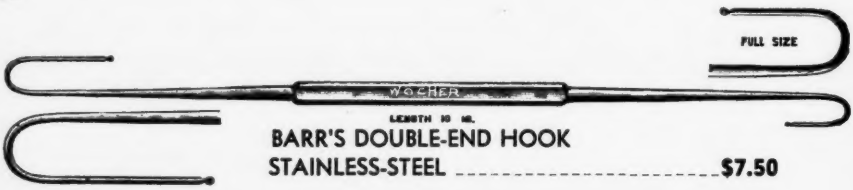


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